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# THE NEPHROTIC SYNDROME IN PREGNANCY

## RENAL BIOPSY STUDIES

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The association of the nephrotic syndrome and pregnancy is not uncommon in the urbanized Johannesburg African, and previous communications[1,2] have dealt with the clinical aspects of this condition. The purpose of the present paper is to report on the renal pathology underlying the syndrome.

### MATERIAL AND METHODS

Seven patients were included in this study, from all of whom renal biopsy material was obtained. These patients constitute 25% of all the pregnant nephrotic patients we have seen to date. All the patients studied satisfied the conventional criteria for the nephrotic syndrome, presenting with marked oedema, proteinuria, hypo-albuminaemia and hypercholesterolaemia, in the absence of hypertension or azotaemia (Table I). All were first seen and diagnosed during pregnancy. Two patients stated that the onset of the oedema was in the first month of pregnancy and 2 said it was in the second month. In 2 cases oedema was first noted 2 years before conception and in 1, 4 months before. Six patients were delivered of full-term, normal infants. The seventh was delivered of twins in the 37th week of pregnancy. One of these twins died of asphyxia following an internal version and breech extraction for a transverse lie. None of the cases was complicated by hypertension (defined as a blood pressure greater than 120/80 mm.Hg) or impairment of renal function during the period of observation. After delivery the full syndrome was still present in all cases. The oedema, however, usually regressed shortly after delivery. In all cases treatment of the oedema consisted principally of bed rest, a high-protein, low-salt diet and chlorothiazide. Steroid therapy was not given.

Percutaneous renal biopsy[3] was performed either during the first half of pregnancy or in the postpartum period. In 2 cases biopsy was done twice, at the 16th week of pregnancy and again after delivery.

Biopsy material was fixed in formol sublimate and sections were cut at 5$\mu$ thickness. Slides were stained with haematoxylin and eosin, the periodic-acid Schiff technique, and elastic-Masson when necessary. Unfortunately there was insufficient material in the biopsy specimens for frozen sections to be examined for fat.

### PATHOLOGICAL FINDINGS

Table II summarizes the main pathological findings in the 7 subjects.

All cases showed essentially similar features, the appearances being those of the Ellis type II or membranous glomerulonephritis. The most striking change was seen in the glomerular basement membrane which showed varying degrees of thickening (Figs. 1 and 2). This appearance was seen focally in some glomeruli, diffusely in others, and affected all or most glomeruli. The capillaries in general presented a rather rigid appearance with reduction in their lumens in some instances. Case 3 showed early necrosis of some glomerular tufts (Fig. 3). There was slightly increased cellularity in the tufts of case 5. Tubular casts were a variable feature, being present in 2 cases. There was slight atrophy of the proximal convoluted tubules in case 3. The latter biopsy was also striking for the large number of foamy macrophages in the interstitial

TABLE I. DATA CONCERNING 7 PATIENTS UNDERGOING RENAL BIOPSY

| *Case* | *Age (yrs.)* | *Parity* | *Serum albumin (g.%)* | *Plasma cholesterol (mg.%)* | *Blood urea (mg.%)* | *Proteinuria (g./l. Esbach)* | *Outcome of pregnancy* | *Time of biopsy* |
|---|---|---|---|---|---|---|---|---|
| 1 | 18 | 0 | 0·8 - 0·9 | 360 - 418 | 14 - 32 | 1·5 - 18 | Full-term normal delivery | (*a*) 16th week of pregnancy<br>(*b*) 3 months post-partum |
| 2 | 25 | 2 | 1 - 1·5 | 305 - 350 | 11 - 18 | 0·5 - 5 | Full-term normal delivery | 2 weeks postpartum |
| 3 | 22 | 0 | 1·2 - 2·2 | 196 - 245 | 13 - 21 | 1·5 - 9 | Full-term normal delivery | 3 weeks postpartum |
| 4 | 24 | 2 | 0·4 | 670 | 17 | 0·5 - 9 | Full-term normal delivery | 18th week of pregnancy |
| 5 | 29 | 5 | 1·8 - 1·9 | 310 - 321 | 10 - 28 | 0·25 - 2 | Twins at 37th week, 1 of whom died of asphyxia | 3 weeks postpartum |
| 6 | 21 | 2 | 1·0 | 540 | 15 | 1 - 6 | Full-term normal delivery | (*a*) 16th week of pregnancy<br>(*b*) 1 week postpartum |
| 7 | 23 | 1 | 0·7 - 1·1 | 265 - 305 | 11 - 16 | 1·5 - 7 | Full-term normal delivery | 1 week postpartum |

TABLE II. RENAL BIOPSY FINDINGS IN 7 CASES OF THE NEPHROTIC SYNDROME IN PREGNANCY

| *Case* | *No. of glomeruli* | *Basement-membrane thickening* | *Capillaries of tuft* | *Tubules* | *Interstitial tissue* | *Vessels* |
|---|---|---|---|---|---|---|
| 1 (*a*) | 5 | Diffuse, affecting every glomerulus | Normal and patent | Granular casts, occasional hyaline droplets in proximal convoluted tubules | Normal | Normal |
| (*b*) | 13 | Diffuse, affecting every glomerulus | Slightly reduced lumens | Granular casts, occasional hyaline droplets in proximal convoluted tubules | Normal | Normal |
| 2 | 5 | Affecting all parts of every glomerulus | Some with reduced lumens | Normal. No casts | Normal | Normal |
| 3 | 7 | Affecting all parts of every glomerulus. Early hyalinization of some glomeruli | Some with reduced lumens | Slight atrophy of proximal convoluted tubules. Occasional hyaline casts | Contains numerous foamy macrophages | Slight thickening |
| 4 | 30 | Affecting all parts of every glomerulus | Some with reduced lumens | Granular degenerative changes of proximal convoluted tubules. Occasional hyaline casts | Single focus of lymphocytic infiltration | Normal |
| 5 | 25 | Affecting all glomeruli, diffuse in some, focal in others | Some with reduced lumens | Normal, occasional casts | Normal | Normal |
| 6 (*a*) | 28 | Affecting all parts of every glomerulus | Some with reduced lumens | No casts. Occasional hyaline droplets | Normal | Normal |
| (*b*) | | Affecting all parts of every glomerulus | Some with reduced lumens | No casts. Occasional hyaline droplets | Normal | Normal |
| 7 | 3 | Affecting all glomeruli diffusely | Some with reduced lumens | Normal | Normal | Normal |

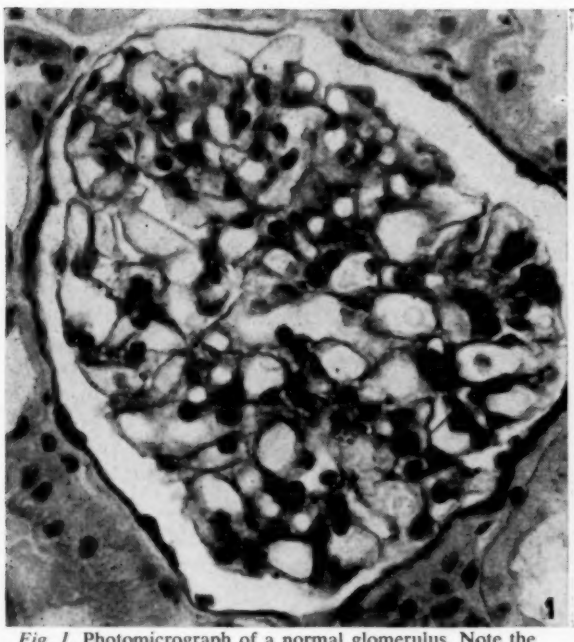

*Fig. 1.* Photomicrograph of a normal glomerulus. Note the delicate basement membrane. (periodic-acid-Schiff × 600).

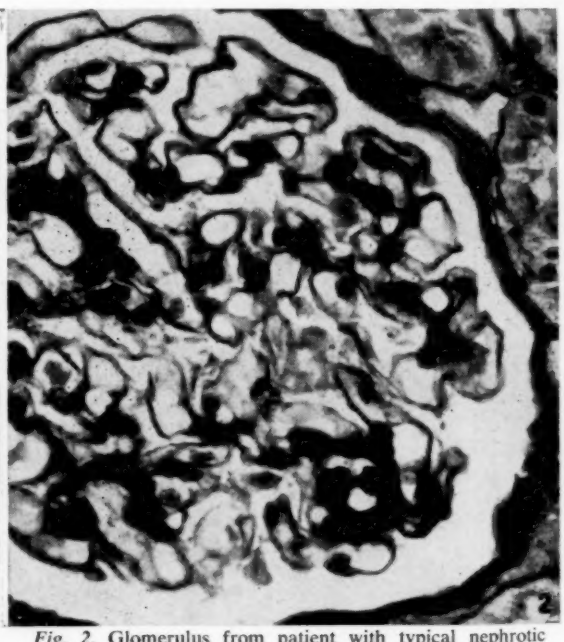

*Fig. 2.* Glomerulus from patient with typical nephrotic syndrome. There is marked, diffuse basement-membrane thickening (periodic-acid-Schiff × 600).

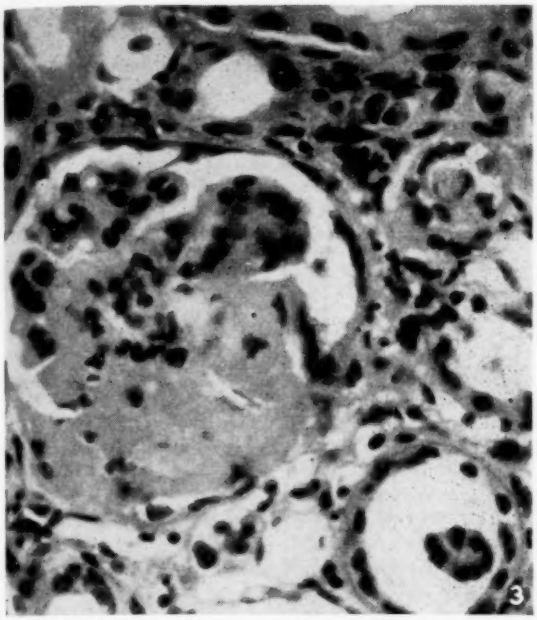

*Fig. 3.* Kidney with more advanced changes showing partial fibrosis of a glomerulus and atrophy of tubules. (haematoxylin and eosin×600).

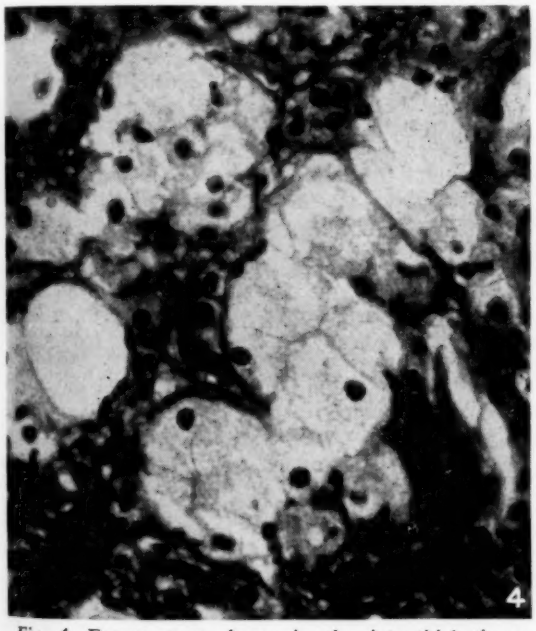

*Fig. 4.* Foamy macrophages in the interstitial tissue (haematoxylin and eosin×600).

tissue (Fig. 4). In all the other subjects the interstitial tissue was normal. In no case was there any significant abnormality in the arterioles or arteries.

## DISCUSSION

Renal-biopsy studies have shown that the nephrotic syndrome may be associated with a variety of renal lesions — membranous glomerulonephritis, proliferative glomerulonephritis, a combination of these, diabetic glomerulosclerosis, systemic lupus erythematosus, amyloidosis, renal-vein thrombosis, nephrosclerosis,[4,5] and nephrocalcinosis.[6] It is therefore of interest that these 7 African patients with the nephrotic syndrome associated with pregnancy all showed the changes of membranous glomerulonephritis (Ellis type II nephritis). The number of cases is admittedly small, but our findings in non-pregnant African nephrotic patients are essentially similar (unpublished observations). Of note also, is the absence of any progression in the renal lesion in the 2 patients in whom biopsy was done at the 16th week of pregnancy and again after delivery.

Previously we have demonstrated that the association of the nephrotic syndrome and pregnancy in the African usually has a favourable outcome,[2] as shown by the cases in this series. By contrast, some British and American authorities have stated that the maternal and foetal prognosis is poor.[7-10] The reasons for this divergence of opinion are obscure. In part it may be due to the inclusion of cases of the syndrome complicated by hypertension or azotaemia, factors, it would be generally agreed, which affect the outcome of pregnancy adversely.

A further reason may be that prognosis in the pregnant nephrotic patient is related to the nature of the underlying renal lesion. Our own observations indicate that, when Ellis type II or membranous glomerulonephritis is associated with pregnancy, the maternal and foetal prognosis is good. The same may not be true of other lesions responsible for the nephrotic syndrome. Renal biopsy studies in a British or American series of pregnant nephrotic patients have not yet appeared, but some idea of the possible findings may be gained from observations in non-pregnant cases. In these it has been shown that a substantial number are due to lesions other than membranous glomerulonephritis.[4,5] Thus about half of the 98 cases of the nephrotic syndrome reported by Kark *et al.*[4] were due to such lesions as diabetic glomerulosclerosis (15 cases), systemic lupus erythematosus (18 cases), proliferative glomerulitis (6 cases), or mixed proliferative and membranous glomerulonephritis (12 cases). Many of these are characterized by a more florid and active form of glomerular disease than pure membranous glomerulonephritis and pursue a more rapidly progressive clinical course. It is possible that such lesions are aggravated by pregnancy but, even if this is not so, their natural rate of progression may be so rapid that, in the course of pregnancy, considerable deterioration in renal structure and function may occur.

## SUMMARY

In 7 African cases of the nephrotic syndrome in pregnancy renal biopsy was performed either during or after pregnancy.

All cases showed the changes of membranous glomerulonephritis. In 2 cases in which biopsy was done at the 16th week of pregnancy and again after delivery, no progression in the renal lesions was observed.

The bearing of these findings on maternal and foetal prognosis is discussed.

We would like to express our thanks to the Superintendent of Baragwanath Hospital for permission to publish; Dr. D. W. P. Lavery, from whose department the clinical material was largely drawn, and the Director of the South African Institute for Medical Research, for facilities granted. Mr. M. Ulrich produced the photomicrographs.

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# STUDIES ON THE FAT-TOLERANCE TEST WITH PARTICULAR REFERENCE TO ISCHAEMIC HEART DISEASE*

I. A. D. BOUCHIER, M.B., CH.B. (CAPE TOWN).

Fat-tolerance tests, which measure the pattern of absorption and removal by the body of ingested fat, were carried out on 219 adult males of the 3 racial groups, Bantu, Coloured, and White, some of whom were hospitalized patients whilst others were ambulant volunteers. Of these, 58 had clinical and electrocardiographic evidence of ischaemic heart disease (IHD) and they formed the test group, whilst the remainder, who were apparently healthy, were designated controls.

*Results*

In patients with IHD the alimentary lipaemia was greater and more prolonged than in control subjects over the 7½ hours of study. There was in addition a greater fasting lactescence of the plasma. An overlap between the test and the control subjects was present, but the differences were significant 6 and 7½ hours after fat ingestion.

The test group was subdivided into 2 sub-groups, dependent upon whether phenindione ('dindevan') was being taken or not. The patients on phenindione, as a group, showed much less lipaemia than the group not on anticoagulant therapy. Twenty-six controls who were completing a course of parenteral penicillin and streptomycin (as antibiotic cover for minor surgery) were compared with those controls who had not received antibiotics. The patients on antibiotics showed much less lipaemia than the controls throughout the period of the test.

No racial differences in the fat-tolerance test were demonstrated. The age of the candidate did not affect the test.

In the 161 controls an analysis was made of those factors thought likely to influence an individual's response to a high-fat meal. Differences in income, diet, alcohol-intake, smoking habits, familial incidence of IHD, height, weight, skin-fold thickness, and serum-cholesterol level did not influence the test. Comparison between the hospitalized subjects and the non-hospitalized group showed that the former manifested a greater lipaemia following the test meal. It was suggested that (1) the overall ill-health of the patients in the hospital, and (2) their lessened activity were responsible for these findings.

*The Intravenous Fat-tolerance Test*

The mechanism of the greater lipaemia in the patients with IHD could arise from defective absorption or removal of fat. By comparing the effect of both oral and intravenous administration of cottonseed oil ('Lipomul IV') in matched pairs, the process of absorption was avoided and the function of removal was studied. The fat, when fed orally, showed the characteristic differences between test and control subjects. When administered intravenously, no differences in the amount of circulating fat, or the rate of removal of the fat from the circulation, were detected between the two groups. These results were interpreted as indicating that differences in the mode of absorption of fat by subjects with IHD, might well be responsible for the differences in fat-tolerance tests.

*Combined Radioactive $^{131}I$-labelled Fat and Butter-fat Tolerance Test*

A comparison between the fat-tolerance test and the $^{131}I$-labelled fat ('triolein') test was performed by their simultaneous administration to 7 patients with IHD and 11 controls. On the following day the fat-tolerance test alone was repeated. The butter-fat tolerance test showed a greater and more prolonged lipaemia in patients with IHD on both days of the test. No significant difference was found between the radioactive tolerance test of the test and the control subjects in the first 24 hours. Significantly increased levels of circulating radioactivity were demonstrated during the second day of the test, i.e. 24—31½ hours after the administration of the 'triolein'. This would appear to be the explanation for the greater fasting plasma lactescence of the subjects with IHD. The experiment also suggested that mobilization of the recently ingested fat from the tissues was not a cause of the increased lactescence in subjects with IHD.

During the course of the above studies, various other aspects of the fat-tolerance test were studied. It was demonstrated:

1. That no increase of lactescence resulted from the ingestion of an isocaloric, fat-free meal.
2. That the fat tolerance of an individual was reasonably constant.
3. That no change in the serum-cholesterol level occurred during the period of the fat-tolerance test.
4. That standing overnight did not influence the lactescence of either plasma or serum.
5. That there were indications that lactescence of plasma differed from that of serum.
6. That a slight rise in the erythrocyte-sedimentation rate occurred during the period of the test.

*Abstract of paper delivered at a meeting of the University of Cape Town Research Forum, Groote Schuur Hospital, Cape Town, on 15 June 1960.

Assistance in the performance of these studies was given by the Ischaemic Heart Disease Research Unit of the Clinical Nutrition Research Unit. This Unit is supported in the University of Cape Town by the South African Council for Scientific and Industrial Research. The investigation was supported in part by a research grant H-3316 (C1) from the National Heart Institute, Public Health Service, USA.

## VAN DIE REDAKSIE : EDITORIAL

### ASPEKTE VAN DIE PROFESSIONELE VERHOUDING TUSSEN SPESIALISTE EN ALGEMENE PRAKTISYNS

By die geleentheid van sy onlangse sitting in Kaapstad is die jongste beskikbare opgawes oor die aantal geregistreerde mediese praktisyns in die land weer, soos gewoonlik, aan die Suid-Afrikaanse Geneeskundige en Tandheelkundige Raad voorgelê vir kennisname en oorweging. Uit hierdie syfers blyk die volgende:

Aan die einde van 1960 was daar altesaam 7,939 geneeshere in die land, waarvan 1,591 geregistreerde spesialiste was. In 1959 was hierdie syfers 7,788 en 1,505 respektiewelik. Dit beteken dus dat daar oor die algemeen ongeveer 1 spesialis teenoor 4 algemene praktisyns was. Gedurende die bespreking van hierdie aspek van die saak het prof. H. W. Snyman, van Pretoria, tereg aangetoon dat die globale verhouding min of meer normaal was.

Die saak is egter nie net 'n kwessie van absolute verhouding nie. Daar is in Suid-Afrika 'n groot wanverdeling van geneeshere met 'n relatiewe groot opeenhoping van spesialiste in die stedelike gebiede. Dit beteken dus dat die verhouding tussen spesialiste in die stede soms nagenoeg 1 : 2 is, teenoor 'n verhouding van 1 : 12 of meer in sekere plattelandse gebiede. Bloot die feit van hierdie wanverdeling verander al die hele saak. Daarby het dr. A. W. S. Sichel aangetoon dat die genoemde syfers op 'n ongesonde tendens dui, byvoorbeeld, dat terwyl die persentasie toename van algemene praktisyns gedurende die afgelope jaar minder as 1 persent was, die toename van spesialiste nagenoeg 6 persent was. As hierdie tendens sou voortduur, en dit wil voorkom of dit wel die geval is, dan sal ons weldra 'n relatiewe sowel as absolute wanverhouding hê tussen spesialiste en algemene praktisyns in die land.

Hierdie hele saak sal vroër of later weer oorweeg moet word, byvoorbeeld in die lig van die vraag of dit nie gewens sou wees om weer terug te gaan na 'n stelsel van suiwer konsulterende spesialiste nie. Ons weet nie wat die antwoord op dié vraag is nie; ons stel maar net die vraag om nadenke te prikkel. Wat ons egter tog wel op 'n praktiese vlak aan die saak kan doen, is om alle middele binne ons vermoë in werking te stel om die goeie gesindheid en bevredigende professionele samewerking tussen spesialiste en algemene praktisyns te probeer bevorder. Om hierdie rede haal ons dan nou ook hier die reëls van die Mediese Raad aan wat spesifiek oor hierdie aspek van die saak handel. Die reëls waarna ons verwys is reëls 11 - 14 van die ‚reëls aangaande die registrasie van spesialiste . . . en die voorwaardes betreffende die beheer van die praktyke van geneeshere . . . wie se spesialiteite geregistreer is'. Reëls 11 - 14 lees soos volg:

‚11. 'n Spesialis mag nie 'n pasiënt van 'n ander praktisyn, hetsy hy 'n spesialis of 'n algemene praktisyn is, oorneem nie behalwe met die toestemming van die betrokke praktisyn. Sodanige toestemming mag nie onredelikerwys teruggehou word nie.

‚12. 'n Spesialis mag nie tuisbesoeke aflê nie behalwe op versoek of met die toestemming van 'n algemene praktisyn.

‚13. 'n Spesialis kan enigeen behandel wat regstreeks na hom gaan vir raadpleging.

‚14. 'n Spesialis wat deur 'n pasiënt geraadpleeg word of wat 'n pasiënt behandel, moet alle redelike maatreëls tref om die samewerking te verseker van die pasiënt se algemene mediese praktisyn of tandarts (d.w.s. algemene tandheelkundige praktisyn), na gelang van die geval.

‚Opmerking. Niks in hierdie reëls mag in enige opsig inbreuk maak op die gebruiklike etiese standaarde met betrekking tot 'n toestand van nood nie.'

Navolging, in die besonderheid, van hierdie reëls sal veel daartoe bydra om die verhouding tussen spesialiste en algemene praktisyns op 'n goeie voet te hou. By 'n konsiderasie van die verhouding tussen mense is reëls egter nie genoeg nie. Gesindheid tel meer as reëls, en ons beroep is op alle praktiserende geneeshere om, soos die meeste van hulle in elk geval doen, op so 'n manier op te tree dat reëls nie eers nodig is nie. Veral in hierdie tye van spanning en drukte waaronder almal, ook geneeshere, leef, kan ons nie genoeg daarop bedag wees om die eer en status van ons as individuele praktisyns en van die mediese professie as 'n geheel, bo verdenking te hou nie.

### MALPRAXIS INSURANCE

The Atlas Assurance Company Limited announce that, in accordance with their policy to provide members of this Association with the widest possible cover for professional liabilities, they have extended their Malpraxis Policy to indemnify policy-holders against damages and costs incurred in the defence of actions for libel, slander, or defamation of character where such actions arise out of professional acts. This additional cover is given without increase in existing premium charges and will be effective immediately. Within the next week or two policy-holders will receive printed endorsements setting out the precise terms of the extension.

The 'Atlas' have also asked us to bring two important points to the attention of our members. These are:

1. *Limits of liability.* Although the Company makes a practice at each renewal date of reminding practitioners to review the limits of liability under their policies, many policy-holders continue to carry limits which may prove completely inadequate for their full protection. The minimum limit of indemnity for which policies are issued is R4,000 for any one claim, but it is obvious that this would not go very far in the event of a serious claim arising, and all practitioners are earnestly requested to give careful thought to a more realistic indemnity. The Company will continue to attach

slips to renewal notices setting out a scale of premiums for various limits of liability up to R50,000 for any one claim, and all that is necessary is to advise the Company if a higher limit is required when an insurance is renewed. If desired, the Company is prepared to quote for a limit in excess of R50,000.

2. *Partnerships.* The Liability Policy provides a personal indemnity to the insured and consequently a separate policy is issued to each individual doctor indemnifying him for his personal acts. Where two or more doctors practice in partnership, therefore, it is essential that each and every partner should carry adequate liability cover with preferably a similar limit of indemnity in each case. It will be clear that if, for example, one of the partners is uninsured and judgment in a civil case is given against him, then the resulting financial loss may well have a serious adverse effect on the partnership as such. Another possibility is that a partnership may be sued for the acts of one of its members and, to overcome this, the Company is willing (provided all partners carry 'Atlas' policies) to endorse the individual policies so that, in the event of a claim against the partnership for the acts of one of them, the protection will apply to those 'innocent' members of the partnership who may be brought into the action, notwithstanding that they themselves were not concerned in the treatment or the cause giving rise to that action.

The local Branch of the Company, through whom your insurance is placed, will be pleased to advise and assist you in this matter of arranging suitable protection for partnerships.

## THE MEDICAL DIRECTORY OF SOUTH AFRICA

The Knox Printing and Publishing Company is to be congratulated on its enterprise in producing a Medical Directory for South Africa. What a wealth of information it contains! There is hardly a facet of medical professional life that is not mentioned in one form or another, and all is subject to easy reference.

It is obvious that the Editor, Dr. Crowhurst Archer, and his staff undertook a monumental task in the first compilation of such a work, and as a profession we should be grateful. Not only will this fulfil a long-felt need, but it is certainly evidence that the medical profession has reached an important milestone in its progress. It is an effort that merits the support of all practitioners so that it may long continue to serve the profession.

The *Medical Directory of South Africa* was published in December 1960, but it has been styled the 1960-61 edition since the information in it was kept right up to date. The price of the Directory is R4.20 and it is obtainable directly from the publishers or from the leading booksellers throughout South Africa.

# AORTO-ILIAC OCCLUSIVE DISEASE*

## A REPORT ON 67 CONSECUTIVE CASES TREATED SURGICALLY

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A recent annotation in the *Lancet*[1] points out that 'over a hundred years have passed since Charcot described the association between intermittent claudication and absent foot pulses; but until quite recently ischaemic pain was commonly misdiagnosed in the early stages, generally as arthritis or a prolapsed disc'.

In reviewing 67 cases of aorto-iliac thrombosis operated upon by us during the past 2 years, we found that a considerable number of the patients appeared at the hospital with 'lumbago and sciatica' as their initial complaint. Many of them joined the unhappy throng of backache sufferers to torment and test the skill of our orthopaedic surgeons, the referring doctors having suspected 'slipped disc', lumbo-sacral strain, sacro-iliac disease, osteoarthritis of the spine, hip or knee joints, or even disorders of the feet with 'back strain'. No less than 18 received orthopaedic treatment for this complaint and many others were referred for physiotherapy.

* Paper presented at the Annual Congress of the South African Orthopaedic Association (M.A.S.A.), Cape Town, 13 October 1960.

### *Low Back Pain*

The clinical diagnosis of 'low back pain' has become a rubbish heap which includes symptoms varying from a trifling and fleeting discomfort to a prolonged and crippling disability, with or without radiation to the legs.[2] Backache is extremely common — it is among the initial complaints of 1 in every 10 patients.[3] Literally thousands of cases are dealt with by a single orthopaedic clinic every year. Its causes are so numerous and diverse that it presents a considerable dilemma to both patient and doctor. It may originate not only from orthopaedic but also from gynaecological, urological, abdominal, neurological, constitutional and even from psychosomatic disorders. In many instances, however, the mechanism initiating the 'low back pain' remains undiagnosed. Such patients are only too frequently referred from consultant to consultant and launched upon a merry-go-round which usually includes the X-ray and physiotherapy departments and is only too well known in hospital practice.

It is part of the purpose of this paper to point out that among these unfortunate victims of 'passing the buck' are to be found sufferers from aorto-iliac thrombosis, patiently

'claudicating' along the corridors, crying for relief from their cramps. This may go on for months, or even years, as is illustrated by the following case history:

*Case 25*

Mrs. R.S., aged 53 years, was referred to the medical outpatient department at Groote Schuur Hospital in July 1958, because of aching in her legs of 6 months' duration. Peripheral vascular spasm was suspected and antispasmodics were prescribed.

Her symptoms became progressively worse, and in June 1959 she returned to the hospital complaining of backache, and pain and fatigue in the calves, thighs and hips on exercise. She also noticed numbness and paraesthesiae of both legs. She was referred to a surgeon who noted that her pedal pulses were absent, but plethysmography showed good pulsations in the toes and an excellent response to reflex body heating. His comment was, therefore, 'minor peripheral vascular insufficiency with well-marked functional overlay', and she was referred to a psychiatrist, who prescribed physiotherapy which was of no avail. In due course she returned to the physician who referred her to another surgeon. His notes read: 'Story definitely not classic claudication; pain occurs at any time, but worse on walking'. He thought the plethysmograph showed satisfactory circulation and found a 'faint dorsalis pedis pulse on the right', and 'crepitus in both knees'. Osteoarthritis of the hips, knees and lumbo-sacral spine was diagnosed, and X-rays of these regions were requested.

The X-rays were normal and she went back to her physician who, now desperate, referred her to the orthopaedic department with the comment: 'This lady complains of pain in her thighs and legs when she walks. I think her peripheral vasculature is impaired, but the vascular clinic reports minimal deficiency'. The orthopaedic surgeon's report was curt, to the point, and emphatic: 'Pain only present on walking. Starts in calves and spreads up to thighs. Goes on resting. No orthopaedic cause of the pain found. Clinically this appears to be a gradually occluding thrombosis of the lower aorta and common iliacs'.

By this time her claudication distance had decreased to 40 yards and she was physically and mentally in great distress. She was referred to a vascular surgeon who found both legs pulseless apart from very weak femoral pulses. He had no hesitation in diagnosing aortic thrombosis which was confirmed on aortography.

In September 1959, aorto-iliac endarterectomy was performed. This was followed by a return of all the pulses in her legs and complete relief of all her symptoms. She has remained fit and well since then and one year later all her pulses are still present.

Others are even less fortunate and their vicious circle may be rudely interrupted by a succession of surgical procedures which only add to their agony. This is illustrated by the following case report:

*Case 48*

Mr. M.S., aged 46 years, fell off a ladder in 1951 and injured his back. A few days later he first experienced pain in the back and hip regions radiating to the right thigh and calf. This pain was brought on by strenuous exercise and relieved by rest. It was associated with weakness of the leg.

Massage and manipulation of his back failed to improve his symptoms, and in 1952, following an X-ray of his spine, he had a 'slipped disc' removed. There was still no improvement and in the same year he had another operation — fusion of the lumbar vertebrae. The graft did not take and the pain in his right leg became worse, coming on after less and less exercise. In 1953 another spinal graft was done and his coccyx was removed. This also failed to relieve the symptoms.

In 1955 he was referred to Groote Schuur Hospital because of suspected 'Buerger's disease'. Inquiry into his symptoms revealed that the pain in his hips and right leg had the features of intermittent claudication, i.e. it came on only after exercise and was always relieved by rest. In addition it was ascertained that since the injury in 1951 he had been impotent, i.e. unable to have a penile erection, for which psychiatric opinion had been sought without avail. On examination it was found that his right leg was cold and somewhat pale with a poor femoral pulse and absent pedal pulses. The left leg appeared normal, but all the pulses were diminished. Bilateral lumbar sympathectomy was performed. After this the right leg became warm, but there was no improvement in his claudication.

In 1956 he had an attack of coronary thrombosis followed by angina pectoris which prevented him from taking much exercise. This gradually improved, however; then his legs started worrying him again.

In December 1958 aortography revealed aorto-iliac occlusive disease, and aorto-iliac endarterectomy was performed. This was followed by a return of all his pulses and complete relief of all symptoms including the backache and impotence.

He remained symptom-free and fit for one year. Then he started developing claudication of his left calf which rapidly increased to grade 3 at 50 yards. This was found to be due to femoro-popliteal thrombosis and was completely relieved by endarterectomy in May 1960. Since then he has been well.

These 2 case reports serve to illustrate that aorto-iliac thrombosis should be kept in mind whenever patients complain of the type of symptoms described.

*Mistakes in Diagnosis*

Before Leriche's description of thrombotic occlusion of the terminal aorta in 1940 (quoted by Leriche and Morel[15]), the condition was not recognized as a clinical entity and mistaken diagnoses were common.[11] Calf claudication due to vascular insufficiency was well known, but the backache, gluteal and sciatic claudication and other symptoms of aorto-iliac occlusive disease were largely unknown. Leriche[18] drew attention to the significance of these symptoms and pointed out that aorto-iliac disease should always be borne in mind in patients with such complaints.

He warned that 'one should never diagnose a "neuritis" or a "polyneuritis" of the lower limbs, unless one has carefully examined the femoral pulses and the oscillometric curve', and stated that 'when a patient complains of severe fatiguability of the lower limbs, or of pain in the thighs on exertion, if the physician finds alterations in the peripheral pulses, the provisional diagnosis of thrombotic obliteration of the aortic bifurcation may be made'.

Since that time the condition has become well-recognized and is usually referred to as 'Leriche's syndrome'. Nevertheless, mistakes in diagnosis are still made simply because the condition is not thought of and clinicians are not aware of a number of pitfalls in the symptomatology which tend to cloud the issue. Such mistakes often cause delay in giving correct treatment and so may lessen the chances of success. This has become particularly important nowadays because the disease is not uncommon (incidence 0·12 - 0·15%[29]) and recent advances in arterial surgery have made it possible to give many patients complete relief from their symptoms. Furthermore, mistakes in diagnosis may also lead to mistaken treatment, including unnecessary surgical operations (Table I).

We hesitate to discuss the formidable list of orthopaedic procedures listed in Table I, but must mention that none of them gave any relief to the patients' symptoms. The operations included radical removal of toenails, Keller's operation, removal of knee cartilages and intervertebral discs, spinal fusion and even excision of the coccyx. However, it is perhaps comforting to know that this

TABLE I. PREVIOUS SURGICAL PROCEDURES PERFORMED FOR SYMPTOMS AMONG PATIENTS IN THIS SERIES

| *Procedure* | *Patients* |
|---|---|
| Lumbar sympathectomy | 11 |
| Stripping of varicose veins | 2 |
| Orthopaedic procedures: | 18 |
| Operations on feet and/or toes | 5 |
| Operations on knees | 2 |
| Spinal and/or hip manipulations | 10 |
| Spinal extension and/or support | 4 |
| Laminectomy and/or spinal fusion | 4 |
| Removal of coccyx | 1 |
| Hysterectomy | 2 |
| Ventral suspension | 1 |
| Appendicectomy | 1 |
| Carotid angiography | 1 |
| Thyroidectomy | 1 |

experience is not limited to Cape Town. There are many reports in the literature of similar mistakes. De Wolfe *et al.*[13] found that, of their cases, 'a significant number were referred as orthopaedic problems'. Bonney[4] reported on 9 patients who had been referred to the Royal National Orthopaedic Hospital for pain in the buttock or thigh caused by aorto-iliac thrombosis. Filtzer and Bahnson[14] found that 13 of 65 patients with aorto-iliac disease presented with low back pain and 'sciatica'. Ten of them had received orthopaedic treatment including removal of intervertebral discs, spinal fusions and exploration of the hip joint.

It is the principal purpose of this paper to direct attention to the clinical features of aorto-iliac disease. The observations are based on a review of 67 consecutive cases treated by us during the past 2 years, in whom the diagnosis was confirmed by aortography and operative exploration. In addition, brief reference will be made to certain aspects of the pathology and pathogenesis of the condition, treatment, and the results of surgery.

It should be noted that these 67 cases constitute only a fraction of patients seen and treated by us for chronic occlusive vascular disease during the same period. An equal number have been operated upon for femoral occlusions, others have had sympathectomies for distal-vessel disease, and many with both proximal and distal occlusions have been treated conservatively.

## PATHOLOGY

### *Situation of the Lesion*

Our cases have been divided into 5 groups as indicated in Fig. 1.

1. *Aortic bifurcation* (7 patients — Fig. 1A). There was partial or complete obliteration of the lower end of the aorta, usually limited to the segment between the inferior mesenteric artery and the aortic bifurcation. The iliac vessels were patent and remarkably normal although the orifices of the common iliac arteries were occluded. Both limbs were affected, but the femoral and popliteal vessels were patent.

2. *Aorta and common iliac vessels* (15 cases — Fig. 1D). Here the disease was also localized, but in addition to the lower aorta *both* common iliac vessels were partially or totally occluded. The disease often extended proximally beyond the inferior mesenteric artery as far as the renal vessels, but in no case was there evidence of renal arterial stenosis. The external and internal iliac vessels were patent, but stenosed at their origins. The femoral and popliteal vessels were patent.

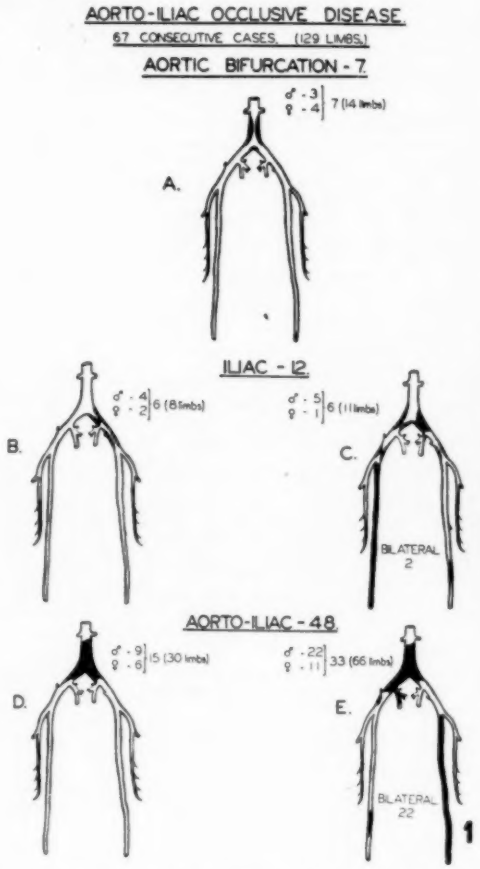


*Fig. 1.* Situation of the lesion: (A) aortic bifurcation, (B) iliac, (C) ilio-femoral, (D) aorto-iliac, and (E) aorto-ilio-femoral.

3. *Iliac* (6 cases — Fig. 1B). The occlusion was bilateral in 2 and unilateral in 4 patients. The occlusion was situated at the common iliac bifurcation in 6 limbs with involvement of the internal iliac artery in 3. In 2 limbs the distal part of the external iliac was affected. The femoral and popliteal arteries were patent in all the patients.

4. *Ilio-femoral* (6 cases — Fig. 1C). This represents more diffuse disease affecting several vessels. In 1 patient only the left common iliac and superficial femoral vessels were involved. In the others the iliac occlusions were bilateral. In 2 of them all the iliac vessels (common, external and internal) were occluded, and in 2 the common and external iliac arteries only. There was bilateral femoral involvement in 2 patients.

5. *Aorto-ilio-femoral* (33 cases — Fig. 1E). This represents very extensive disease affecting several vessels on both sides in all cases. The pattern varied from involve-

ment of the aorta and 1 common iliac and 1 femoral artery; to extremely diffuse involvement of the aorta up to the renal vessels and above, both common iliac arteries, both external iliacs, both internal iliacs and both superficial femorals. The internal iliacs were occluded in 6 patients and both femorals were affected in 22 patients.

*Nature of the Lesion*

*Atheroma.* (Fig. 2). Although not all writers have accepted the atheromatous origin of aortic thrombosis,[29] our findings support the view that thrombosis is usually superimposed on underlying atheroma.[8,10,29] In the vast majority of cases there was obvious evidence of extensive atheroma, with or without calcification of the vessels, and in 4 of the patients fresh, organizing clot was found superimposed on a localized atheromatous plaque. The fatty acids in plaques removed at operation have been fractionated and the pattern closely resembles that found in the plasma. This is the subject of a further report.

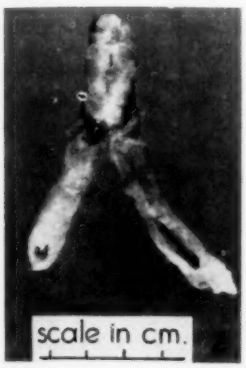


*Fig. 2.* Atheromatous plaque removed from aortic bifurcation and common iliac vessels.

*Buerger's disease.* A striking feature in some of the cases was an extensive periarteritis. This was most often found in occlusions of the external iliac artery and in patients with a recent onset of symptoms. In 2 patients portions of such vessels were excised and histological examination showed underlying atheromatous changes. In at least 3 of the patients a previous diagnosis of Buerger's disease had been made because of the development of peripheral arterial disease at an early age. These men were aged 30, 40 and 46 years when direct arterial surgery was undertaken, and in all of them diffuse artherosclerosis was found, but no evidence of Buerger's disease.

*Trauma.* In 3 of our patients symptoms commenced immediately after severe injuries to the back and limbs. Trauma might have been a factor in precipitating the thrombosis, but all of them had diffuse atherosclerosis. Others[19,29] have also drawn attention to the possible rôle of trauma.

*Embolism.* In only 1 of the patients was there a history of myocardial infarction immediately before the onset of symptoms.

In this case the thrombosis might have been the result of embolism with retrograde clotting into the aorta. However, none of our patients suffered from mitral stenosis and none of those who gave a history of sudden onset had evidence of cardiac irregularities. On the other hand, the possibility of embolization from more proximal aortic atheromatous lesions still exists and is being investigated further.

## RACE, SEX AND AGE DISTRIBUTION

Of the patients, 62 were European and 5 were Coloured; there were no Bantu (Table II). The population served by Groote Schuur Hospital is made up of 310,000 European, 350,000 Coloured and 107,000 Bantu persons and the admissions during 1959 were 10,000 European, 10,000 Coloured and 2,000 Bantu. It would thus appear that there is a significant racial difference in the incidence of the disease and this is being investigated further.

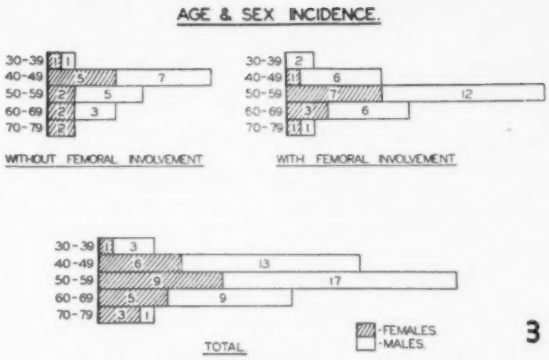


*Fig. 3.* Incidence related to sex and age.

There were 43 male and 24 female patients (Fig. 3). This is in keeping with recent reports by others[29] and

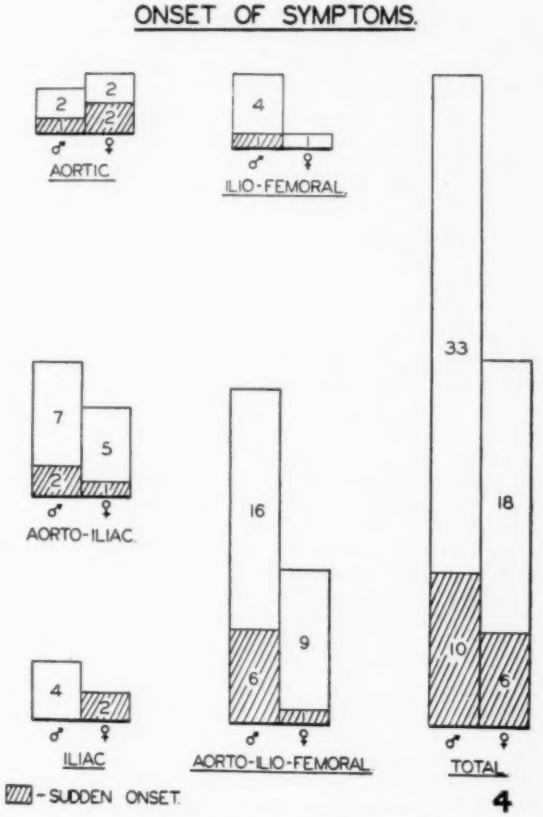


*Fig. 4.* Onset of symptoms (see text).

TABLE II. RACIAL INCIDENCE OF 67 CASES OF AORTO-ILIAC DISEASE

| | *European* | *Coloured* | *Bantu* |
|---|---|---|---|
| Population .. .. | 310,000 | 350,000 | 107,000 |
| Annual admissions .. | 10,000 | 10,000 | 2,000 |
| Aorto-iliac disease .. | 62 | 5 | — |

contrary to the findings of earlier writers, including Leriche[18] who believed that the disease occurred almost exclusively in males. It has been suggested that the pathogenesis of the disease in women differs from that in males.[28,29] In our series there does seem to be a higher incidence of localized aortic or aorto-iliac disease in female patients, viz. in 10 (42%) of the 24 women and in 12 (28%) of the 43 men the disease was limited to this area. On the other hand, iliac disease without aortic involvement was much less common in females (3 cases) than in males (9 cases). It should be noted that 19 of the 24 women, including all but 1 of those with localized occlusions, were parous.

The youngest patient was a male aged 30 years, and the oldest a female of 74 years. The age distribution given in Fig. 3 shows that the peak incidence for both males and females is during the sixth decade. Localized iliac occlusions were found particularly in young males (average age 46 years). More striking, however, was the occurrence of localized aortic or aorto-iliac disease in young females. One old lady of 72 who looked remarkably young for her age exaggerates the average age of this group which is 47 years — 1 patient was only 33 and 5 were in the 'early forties'. This also suggests that the pathogenesis in females differs from that in males.

ONSET AND DURATION OF SYMPTOMS

In 16 of the patients the onset was sudden (Fig. 4). In 3 of them trauma was apparently a precipitating factor and in 1 embolization from a mural thrombus following a myocardial infarct might have been responsible. It may be significant that 3 of the 10 females with localized aortic and aorto-iliac occlusion and both females with localized iliac disease had a sudden onset of symptoms.

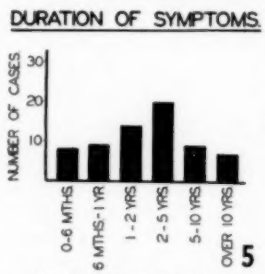


*Fig. 5.* Duration of symptoms.

The length of history varied from 2 weeks to 18 years, with an average of 3½ years (Fig. 5). In general, the duration tended to be shorter in patients suffering from localized occlusions, but even among these there were patients who had had symptoms for up to 10 years. In the majority of cases the symptoms increased slowly, but progressively. In some there was, at first, rapid progression from stage 1 to stage 3 claudication over a period of months, and then very slow progress. In 3 patients with aorto-ilio-femoral occlusions there was a history of sudden deterioration after a long static period, no doubt due to the later onset of femoral thrombosis.

(*To be continued*)

# THE INCIDENCE OF HYPOCHROMIC ANAEMIA IN PREGNANT BANTU WOMEN

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In a recent investigation of the mean haematological values in pregnant European, Cape Coloured and Bantu women in Cape Town, Lanzkowsky[1] came to the conclusion that the incidence of hypochromic (iron-deficiency) anaemia was high in the three main racial groups. Previous results published by Gerritsen and Walker,[2] and latterly by Metz,[3] have indicated the converse, namely, that iron-deficiency anaemia is very uncommon in pregnant Bantu women. Both these investigations were carried out on subjects in the neighbourhood of Johannesburg, which may account in part for the difference.

In view of these conflicting reports and the importance of the problem, the present study was undertaken.

MATERIAL AND METHODS

Altogether, 194 pregnant Bantu women who attended the prenatal clinic at the Lady Selborne Mission Hospital near Pretoria were investigated. The subjects were therefore living at an altitude of approximately 4,500 feet above mean sea level. The cases were not selected and were consecutive patients who attended the clinic during a six-week period. In addition, 38 non-pregnant, clinically normal Bantu nurses, and 32 non-pregnant Bantu women who attended the same clinic as the pregnant women, were investigated. The pregnant and non-pregnant Bantu women were mostly domestic servants and can be considered as a socio-economically and educationally less-privileged group than the nurses.

Haemoglobin was determined as oxy-haemoglobin according to the method of King *et al.*[4] The red-cell count, packed-cell volume (PCV), and erythrocyte sedimentation rate (ESR), were determined according to standard methods.[5] In addition to these determinations blood smears were prepared and examined microscopically.

RESULTS

The pregnant women were divided on the basis of their length of pregnancy, and fell into 3 groups according to the 3 trimesters of pregnancy, viz. less than 20 weeks, 21 - 30 weeks and 31 - 40 weeks. The results of the haematological determinations on these patients are presented in Table I.

Two of the pregnant women (1·03%) had haemoglobin levels below 10 G. per 100 ml. The haematological values

TABLE I. HAEMATOLOGICAL VALUES OF PREGNANT BANTU WOMEN

| *Stage of pregnancy* | *No. of cases* | *Haemoglobin (G. per 100 ml.)* | *Red-cell count (mill./c. mm.)* | *PCV (%)* | *MCHC (%)* | *MCV (cμ)* | *ESR (mm./hr.)* |
|---|---|---|---|---|---|---|---|
| <20 weeks .. .. | 41 | 13·05* (11·0 - 16·7)** | 4·21 (2·90 - 5·50) | 36·9 (33 - 46) | 34·6 (33 - 39) | 87·3 (71 - 113) | 36·7 (14 - 67) |
| 21 - 30 weeks .. .. | 60 | 12·40 (11·0 - 14·3) | 3·98 (2·97 - 5·10) | 35·3 (33 - 44) | 34·4 (33 - 39) | 87·6 (69 - 113) | 41·9 (14 - 59) |
| 31 - 40 weeks .. .. | 93 | 12·40 (8·2 - 15·3) | 3·99 (2·50 - 5·20) | 37·4 (30 - 45) | 32·6 (27 - 41) | 94·4 (66 - 138) | 42·3 (12 - 53) |

PCV = packed-cell volume, MCHC = mean corpuscular haemoglobin concentration, MCV = mean corpuscular volume, ESR = erythrocyte sedimentation rate.
* Average value.
** Range.

of these 2 patients (A and B), both of whom were 38 weeks pregnant, were as follows:

| | *A* | *B* |
|---|---|---|
| Haemoglobin (G. per 100 ml.) | 8·9 | 8·2 |
| Red-cell count (mill./c.mm.) | 3,010,000 | 3,680,000 |
| PCV (%) | 30 | 30 |
| MCHC (%) | 29 | 27 |
| MCV (cμ) | 93 | 100 |
| ESR (mm./hr.) | 63 | 49 |

Hypochromic red cells were present in the peripheral blood smears of both patients.

The results obtained for the whole group of women were analysed statistically in order to determine whether significant differences occurred in the 3 trimesters of pregnancy (Table II).

The results presented in Table II show that the haemoglobin level decreased significantly from the early stages of pregnancy to the mid-term period and thereafter showed no significant change. A significant *increase* in the PCV occurred from the mid-term period to the end of pregnancy.

TABLE II. PROBABILITY LEVELS (TWO-TAILED)* FOUND IN COMPARISONS BETWEEN GROUPS OF PATIENTS AT DIFFERENT STAGES OF PREGNANCY

| | *Periods compared (weeks)* | | |
|---|---|---|---|
| | *< 20 vs. 21 - 30* | *21 - 30 vs. 31 - 40* | *< 20 vs. 31 - 40* |
| Haemoglobin .. .. | P < 1% | P > 5% | P < 0·6% |
| Red-cell count .. .. | P > 5% | P > 5% | P > 5% |
| Packed-cell volume .. | P > 5% | P < 0·1% | P > 5% |

* A probability level P (two-tailed) of 1% or lower should be regarded as indicating a significant difference.

In order to determine whether the number of their previous pregnancies had had any influence on the haematological values of these women, all the women were divided into groups according to the number of previous pregnancies (Table III). When the values found for the group of patients with no previous pregnancies were compared with those for the patients who had had 5 or more pregnancies, it became clear that a significant decrease had occurred in the PCVs of the multiparous group of patients ($P < 0{\cdot}5\%$, two-tailed). The haemoglobin levels and the red-cell counts of these two groups showed no significant differences ($P > 5\%$, two-tailed).

The results of the haematological determinations on 38 non-pregnant Bantu nurses and 32 Bantu women are presented in Table IV. It can be seen that neither the nurses nor the Bantu women had haemoglobin levels below 10 G. per 100 ml.

In Table V the haemoglobin, red-cell count, and haematocrit values of the Bantu nurses and non-pregnant Bantu women are compared with those of the whole group of 194 pregnant women.

These results (Table V) indicate that the haemoglobin, red-cell count, and haematocrit values of both the pregnant and non-pregnant Bantu women were significantly lower than those of the more privileged nurses. However, there were no significant differences between the haematological values of the pregnant and non-pregnant sections of the less-privileged group.

## DISCUSSION

In the present study of 194 pregnant Bantu women, 2 (1·0%) had haemoglobin levels below 10 G. per 100 ml. Metz[3] investigated 147 pregnant Bantu women who attended the antenatal clinic at Baragwanath Hospital. An examination of his results shows that in no case was the haemoglobin level below 10 G. per 100 ml; the lowest being 10·6 G. per 100 ml.

Gatenby and Lillie,[6] citing the work of Dieckmann and Wegner,[7] regarded a haemoglobin concentration of 10 G. per 100 ml. for pregnant women as the lowest value which can be ascribed to hydraemia. If this standard is accepted, the incidence of anaemia in pregnant Bantu women is extremely low compared with that found in other population groups elsewhere in the world. In their study of 4,314 pregnant women in Dublin, Gatenby and Lillie[6] found that 1,027 (24%) had a haemoglobin value of less than 10 G. per 100 ml. Giles and Burton[8] also found a relatively high incidence of anaemia in pregnant women in North Staffordshire. Of the 1,479 patients investigated, 978 (66%) had haemoglobin levels below 80% (11·81 G. per 100 ml.), while in 217 cases the level was lower than 69% (67% = 10 G. per 100 ml.). In an investigation of haemoglobin values in 4,090 subjects in India and Ceylon and 3,020 in Africa, Foy and Kondi[9] found that 'anaemia was widespread and severe, particularly in pregnant women and mostly of the iron-deficiency type'. All these results are in marked contrast to the low incidence found in pregnant Bantu women.

In his investigation, Metz[3] found that the haemoglobin level and PCV decreased significantly from the early stages of pregnancy to mid-term. In the present study similar changes were found in the haemoglobin values. However, the PCV showed no significant decrease. A significant *increase* was in fact found from the mid-term period to

TABLE III. INFLUENCE OF PREVIOUS PREGNANCIES ON THE HAEMATOLOGICAL VALUES OF PREGNANT BANTU WOMEN

| *No. of previous pregnancies* | *No. of cases* | *Haemoglobin (G. per 100 ml.)* | *Red-cell count (mill./c. mm.)* | *PCV (%)* | *MCHC (%)* | *MCV (cμ)* | *ESR (mm./hr.)* |
|---|---|---|---|---|---|---|---|
| Nil .. .. .. | 47 | 12·46* (11·0 - 15·3)** | 4·13 (2·99 - 4·98) | 36·7 (33 - 44) | 34·1 (33 - 42) | 88·8 (55 - 105) | 40·1 (17 - 67) |
| 1 .. .. .. | 52 | 12·48 (8·2 - 16·7) | 4·04 (3·01 - 5·50) | 35·6 (30 - 46) | 34·8 (27 - 41) | 89·2 (71 - 120) | 39·5 (17 - 63) |
| 2 .. .. .. | 28 | 12·20 (10·2 - 13·7) | 3·85 (3·39 - 3·75) | 36·0 (32 - 41) | 33·9 (31·8 - 38) | 93·4 (75 - 117) | 39·2 (12 - 52) |
| 3 .. .. .. | 23 | 12·76 (10·5 - 15·0) | 3·89 (2·67 - 4·89) | 37·0 (32 - 42) | 34·4 (32 - 41) | 97·2 (73 - 138) | 41·8 (27 - 60) |
| 4 .. .. .. | 22 | 12·58 (10·0 - 14·6) | 3·99 (3·56 - 4·60) | 36·6 (30 - 43) | 34·3 (33 - 41) | 91·5 (76 - 107) | 41·1 (17 - 58) |
| 5 or more .. .. | 22 | 12·09 (10·2 - 14·0) | 3·88 (2·72 - 4·70) | 34·3 (31 - 40) | 34·8 (32 - 41) | 88·9 (73 - 136) | 44·7 (24 - 58) |

For key see Table I.
* Average value.
** Range.

TABLE V. COMPARISON BETWEEN HAEMATOLOGICAL VALUES OF NON-PREGNANT BANTU NURSES AND WOMEN AND THOSE OF PREGNANT BANTU WOMEN

| *Group* | *Subjects* | *Haemoglobin (G. per 100 ml.)* | | *Red cell count (mill./c. mm.)* | | *PCV (%)* | |
|---|---|---|---|---|---|---|---|
| | | *Average* | *SD* | *Average* | *SD* | *Average* | *SD* |
| A | Non-pregnant nurses (38) .. .. .. | 13·34 | 1·64 | 4·70 | 0·34 | 43·0 | 4·0 |
| B | Non-pregnant women (32) .. .. .. | 12·10 | 1·40 | 4·03 | 0·55 | 38·0 | 3·3 |
| C | Pregnant women (194) .. .. .. | 12·5 | 1·26 | 4·06 | 0·24 | 36·6 | 17·0 |

| *Comparison of groups* | *P-values (two-tailed*)* | | |
|---|---|---|---|
| A - B | 0·1% | 0·01% | 0·01% |
| B - C | 13·4% | 30·2% | 4·40% |
| C - A | 0·5% | 0·006% | 0·006% |

PCV = packed-cell volume, SD = significant difference
* Significant P-values (< 1%) are underlined.

TABLE IV. HAEMATOLOGICAL VALUES OF NON-PREGNANT BANTU NURSES AND BANTU WOMEN

| | *Non-pregnant Bantu nurses* | | *Non-pregnant Bantu women* | |
|---|---|---|---|---|
| | *Average* | *Range* | *Average* | *Range* |
| Haemoglobin (G. per 100 ml.) .. .. | 13·34 | 10·9 - 16·6 | 12·1 | 10·3 - 14·5 |
| Red cell count (mill./c. mm.) .. | 4·70 | 4·0 - 5·8 | 4·03 | 3·9 - 5·3 |
| PCV (%) .. .. | 43·0 | 39 - 47 | 38·0 | 37 - 45 |
| MCHC (%) .. | 31·0 | 30 - 36 | 32·0 | 30 - 34 |
| MCV (cμ) .. .. | 91·5 | 75 - 100 | 90·5 | 88 - 105 |
| ESR (mm./hr.) .. | 10·0 | 2 - 15 | 22·3 | 3 - 35 |

For key see Table I.

the end of pregnancy. In their investigations, Gerritsen and Walker[2] found that the haematological values remained remarkably constant in a group of pregnant Bantu women during the course of pregnancy. Before these conflicting results can be fully explained, a more extensive study of the problem, including plasma-volume determinations, liver-function tests, and serum-iron studies will have to be undertaken. Such a study is in progress.

The fact that the haematological values of the pregnant women were lower than those of non-pregnant nurses, while no significant differences were found between the values for the pregnant women and those for a socio-economically and educationally similar group of non-pregnant women, indicates the danger of comparing groups which are not strictly comparable in all respects. Complete data on the dietary habits, medical background and nutritional status of the three groups of women would be needed before any conclusions about the possible influence of pregnancy on the haematological values of Bantu women could be regarded as justified.

The present study was not planned to investigate possible factors which might have been responsible for the extremely low incidence of hypochromic anaemia in pregnant Bantu women. In this connection Walker and Arvidsson[10] have suggested that the high iron intake of the Bantu may be of significance. In order to clarify the situation it is obvious that more complete investigations, including accurate estimations of iron intake, will have to be made.

## SUMMARY

Preliminary haematological studies were carried out on 194 pregnant and 70 non-pregnant Bantu women. According to accepted standards, only 2 of the pregnant women showed frank anaemia of the hypochromic type. This incidence of 1·0% is much lower than that reported elsewhere in the world.

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# MID-SYSTOLIC CLICKS

J. V. O. REID, B.M., M.R.C.P., *Department of Medicine,* University of Natal, and King Edward VIII Hospital, Durban*

The multiple aetiology of heart sounds that occur during systole has been recognized in recent years, and in particular a differentiation has been made between those of arterial or semilunar-valve origin (including the commonly encountered ejection clicks), those of atrial origin in atrioventricular dissociation, and those of extracardiac origin. It is with this last variety that the present paper is concerned.

Extracardiac systolic sounds are themselves of multiple aetiology, and their possible causes have been listed by Reid and Humphries.[1] In general, some of these sounds can be clearly recognized to depend on extracardiac features such as a left pneumothorax or mediastinal emphysema. However, a large group remains which was considered extracardiac by Gallavardin,[2] and subsequently by most authors[3-9] although no extracardiac cause was consistently found. The sound in this group was called the mid-systolic click by Wolferth and Margolies,[5] who reviewed the literature. Several series of cases have been recorded.[2,4,5]

## CASE RECORDS

Eight patients have been seen in whom mid-systolic clicks were heard and recorded phonocardiographically, using a Sanborn 'twinbeam cardiette'. A simultaneous lead II tracing of the electrocardiogram was obtained, and in any case where there could be doubt regarding the identification of the second sound, an indirect carotid-artery tracing was made. In 3 cases the phonocardiogram showed that the click was followed by a murmur continuing to the second sound, the murmur being inconstantly present in 1 case. In 3 further patients the click was unaccompanied by a murmur; while in the remaining 2 cases the click occurred during a pansystolic murmur which, in some cycles, was exaggerated following the click. Details of the 8 cases are given in Table I, and the phonocardiograms are shown in Figs. 1 - 8.

In 7 cases the features of the mid-systolic click were similar to those described in the literature. It was heard best in the tricuspid area or just medial to the mitral area and was usually a single sound, but in 1 case it was clearly double (Fig. 7). The intensity varied slightly with respiration and according to whether the patient was lying, sitting or standing. Although the change with posture and respiration was constant for any one patient, there was no constancy from patient to patient, as was shown by Johnston.[4] The position of the sound in systole in these 7 cases was constant as judged clinically, but on the phonocardiogram it could be shown to vary by as much as 0·03 seconds in its relation to the second sound. This variation could clearly be shown in 4 cases to be an increase in the interval between click and second sound on inspiration. In the other 3 cases under consideration, the phonocardiographic interpretation was made difficult by the breath sounds. The relation to the first sound and to the R wave of the electrocardiogram was, in contrast, inconstant.

In the remaining patient (case 5, Fig. 5) the click varied rapidly, unpredictably and very considerably in its position in systole, being anywhere from very early to mid-systole, but never later than two-thirds of the interval between the first and second sounds. It occurred during a pansystolic murmur which in some cycles was accentuated following the click (Fig. 5, first cycle).

## DISCUSSION

The reasons for considering the mid-systolic click to be extracardiac are: the demonstration of pleuropericardial adhesions in 3 cases by Gallavardin,[2] without any intracardiac cause being found; the variability of intensity with respiration and position of the patient;[4,7-9] the presumption that no valve movements take place during systole;[7,9] the variability from cycle to cycle of the time between the R wave of the electrocardiogram and the click;[4] and the character of the sound itself.

Several authors have recorded systolic murmurs that begin with this click. Minhas and Gasul[6] state that the click may or may not precede a systolic murmur, and they record one that began at a systolic murmur and one that occurred in the middle of a systolic murmur. McKusick *et al.*[6] illustrate 2 clicks which originated mid-systolic murmurs. Leatham[7] shows a phonocardiogram in which a loud systolic murmur extended from the click to the second sound; Johnston[4] describes a similar case. Bleifer *et al.*[9] show a phonocardiogram of one such case and state that 'not uncommonly, a systolic murmur originates with a (mid-systolic) click, and extends to the second sound and occasionally into diastole'.

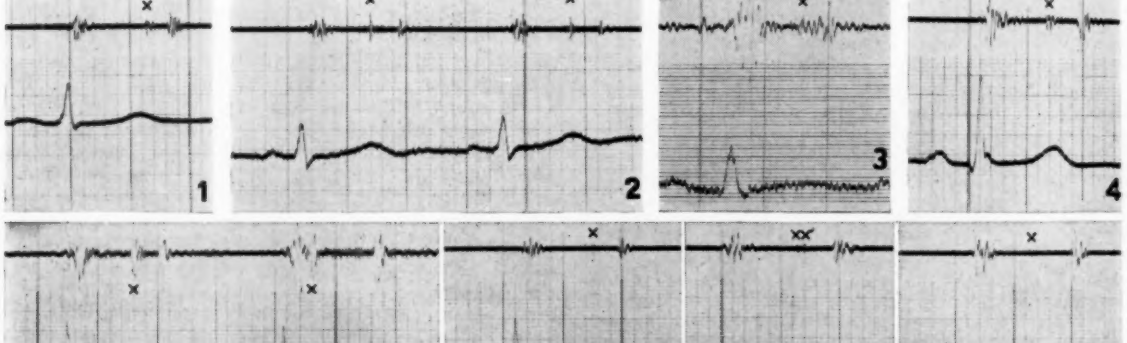
Association with a murmur provides reasonable evidence of an intravascular or intracardiac origin. Because the click is characteristically heard best just within the mitral area or in the tricuspid area, and because in 3 of the present cases a murmur began with the click and continued to the second sound (implying a ventriculo-atrial regurgitation), it seems that the click may be of atrioventricular valve origin in some cases. It does not seem illogical to assume that the valves can be competent at an early stage of systole and become incompetent later, since the design of the atrioventricular valves is unique: the apposition of the cusps is maintained *via* chordae by muscles which themselves are attached to the moving part of the pump, i.e. the ventricular muscle. The papillary muscles contract in systole and so apposition of the cusps is maintained. It is conceivable that a disturbance of this action, for example, by insufficient contraction of a papillary muscle, or by elongation of a chorda, may result in a slackening of the chorda in mid-systole. At this stage the high-pressure gradient across the valve might be expected to snap a slack chorda taut, producing a sound. The attached cusp might then still be competent, producing no murmur; or incompetent, with the production of a murmur that should extend to the second sound or into

* Now Department of Physiology.

TABLE I. FINDINGS IN 8 PATIENTS WITH MID-SYSTOLIC CLICKS

| *Case* | *Race* | *Sex* | *Age* | *History* | *Findings* | *ECG* | *Chest radiograph* | *Comment* |
|---|---|---|---|---|---|---|---|---|
| 1. S.K. | Indian | F | 18 | Incidental findings. No symptoms | MSC in MA followed by murmur | Normal | Normal | |
| 2. Z.K. | Indian | F | 25 | Rheumatic fever, aged 10. Dyspnoea and palpitations for 2 years. On digitalis | Slight LV+. MSC in TA followed by murmur | Digitalis effect | Not done | |
| 3. J.B. | European | F | 49 | Bone pains for 2 years | Classical myelomatosis. Uraemia. Apical and parasternal cardiac thrust. First sound accentuated. B.P. 120/70 mm. Hg. MSC in MA followed inconstantly by murmur | Normal | General cardiomegaly | Diagnosis of amyloid heart not proved |
| 4. J.R. | Indian | M | 19 | Intermittent epigastric pain relieved by food, for 2 months | MSC in MA during a pansystolic murmur | Normal | Normal | Probable peptic ulcer |
| 5. M.M. | African | F | 12 | Weakness of right hand | No neurological abnormality. Slight LV+. MSC in MA during a pansystolic murmur. No evidence of active rheumatic fever | Normal | LV+ | |
| 6. S.P. | Indian | M | 15 | Flitting polyarthralgia | Acute rheumatic arthritis. MSC in TA. No other clinical evidence of active carditis | Q-Tc 0.463 seconds | Normal | |
| 7. V.P. | Indian | F | 14 | Flitting polyarthralgia | Acute rheumatic arthritis. MSC in TA. No other clinical evidence of active carditis | Not done | Normal | Patient seen 1 month before for iron-deficiency anaemia; had no MSC then |
| 8. L.G. | Indian | F | 30 | Incidental finding. Slight dyspnoea on exertion; on close questioning, not considered significant | Depressed sternum. MSC in MA | Normal | Normal | |

MSC = mid-systolic click, MA = mitral area, TA = tricuspid area, LV = left ventricle, + = enlargement.




*Figs. 1-8.* In all figures the phonocardiogram is shown as the upper tracing, lead II of the electrocardiogram as the lower tracing. Except in Fig. 3, logarithmic phonocardiograms are reproduced; in Fig. 3 the phonocardiogram is stethoscopic. The mid-systolic click is denoted by 'X' against the phonocardiogram and by MSC in the legends. Large divisions = 0·2 seconds, small divisions = 0·04 seconds.

*Fig. 1.* Case 1. MSC followed by vibrations continuing to the second sound.

*Fig. 2.* Case 2. MSC followed by a murmur and just preceded by a few vibrations.

*Fig. 3.* Case 3. MSC followed by a murmur continuing to the second sound.

*Fig. 4.* Case 4. MSC in the course of a pansystolic murmur.

*Fig. 5.* Case 5. MSC changing position in systole and occurring during a pansystolic murmur which appears accentuated following the MSC in the first cycle. Breath sounds partly account for the apparent loudness of the murmur in the second cycle, and entirely for the vibrations following the second sound of this cycle.

*Fig. 6.* Case 6. MSC unaccompanied by any murmur.

*Fig. 7.* Case 7. Double MSC unaccompanied by any murmur. The electrocardiogram was not recorded during this cycle, which was taken with the patient sitting, due to electrical interference.

*Fig. 8.* Case 8. Soft MSC unaccompanied by any murmur.

diastole as far as mitral or tricuspid re-opening, as is the case in ordinary mitral and tricuspid incompetence. In accordance with this explanation a mid-systolic click might be termed a 'chordal snap'.

Such a situation does not necessarily imply any visible pathological change in the valve, nor any significant resultant myocardial hypertrophy. However, it would be expected that pathological change in the chordae tendineae, papillary muscles, the valve cusp, or the ventricular muscle would predispose to the occurrence of a mid-systolic click, and be found especially in cases where there is a murmur. It will be observed that, in the 5 cases where systolic murmurs were present, cardiac abnormality was detected in 3. In the 3 cases without a murmur, cardiac abnormality was not detected in any, but was conceivably present in 2 (cases 6 and 7) where acute rheumatic arthritis was present.

No radiological evidence of pleural or pleuropericardial adhesions was found in the 7 patients examined, a finding similar to that of Johnston[4] who observed tenting of the diaphragm in only 1 of 15 cases.

SUMMARY

Mid-systolic clicks can be ascribed in some cases to definite causes such as a small pneumothorax; in many cases no cause is discoverable and the sound is presumed to arise from pleuropericardial adhesions.

In 8 patients mid-systolic clicks have been heard and recorded. In 3 the click was followed by a murmur that continued at least to the second sound, in 2 it occurred during a murmur that continued to the second sound. Similar cases of mid-systolic clicks initiating a murmur ending at the second sound have been described in the literature and have been ascribed similarly to pleuropericardial adhesions.

It is argued that a possible explanation of this mid-systolic click is a snapping taut of a chorda tendinea during the later high-pressure phase of ventricular systole which, when resulting in mitral incompetence, is followed by a murmur that continues, like the murmur of mitral incompetence, at least to the second sound. The term 'chordal snap' is proposed for mid-systolic clicks in which this explanation is thought operative.

I thank Dr. N. A. Rossiter for permission to study and include one patient, and Dr. S. Disler, Medical Superintendent of King Edward VIII Hospital, for facilities.

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## FIRST SURGICAL OPERATION UNDER AN ANAESTHETIC IN SOUTH AFRICA

R. T. S. LOUTTIT, F.R.C.S. (EDIN.), *Surgeon, Basutoland*

On 16 October, 1846 at the Massachusetts General Hospital, USA, the first major surgical operation under an anaesthetic was successfully performed. The surgeon was Dr. J. C. Warren, but the man who introduced ether anaesthesia to surgery was Dr. William Morton, a Boston dentist. The world was not generous to Dr. Morton, and he died at the early age of 49.

News of the epoch-making discovery reached South Africa in the first half of 1847. On 19 June a report appeared in the South African press describing an operation under the new anaesthetic performed by Dr. W. G. Atherstone, a practitioner in Grahamstown.

A second report followed in which the details of the operation and the mode of giving the anaesthetic were published. The following is an extract of the second report:

The patient was a senior government official in the town of Albany. He had suffered an attack of 'erysipelas' in the calf of one of his legs, and this had proceeded to gangrene resulting in severe contracture at the knee joint with extensive callous ulceration, making the limb useless.

After making several experiments with wide-mouthed bottles and rubber tubes, the patient was satisfied about the powerful effects of ether vapour and he consented to have the limb amputated.

The operation was performed on Wednesday, 16 June by Dr. W. G. Atherstone, who wrote as follows:

'I amputated the thigh six inches above the knee. I was assisted by my father, Dr. J. A. Atherstone, and two other surgeons from the 91st Regiment. After ten or twelve inhalations of the ether the patient pinched himself to ascertain what degree of insensibility there was. He continued inhaling a short time longer, and then said "I am drunk enough now—you may begin". The tourniquet was immediately tightened and at the same instant the first plunge of the knife was effected without the least action or sign of suffering on the part of the patient who, at this stage, appeared quite unconscious, and continued inhaling the ether. Two of the assistants removed their hands altogether from the patient's limbs, finding not the slightest resistance. At the second incision, which divided the large nerves and vessels, he uttered an involuntary shriek although not the slightest movement was perceptible, or other symptom of pain. As soon as the leg was off, the bottle was removed; the patient becoming talkative and even humorous as he gradually recovered from the stupefying effects of the ether. The time during which the ether was inhaled was about three minutes.

'He has since stated that the impression on his mind was that he was present at an amputation performed upon some other person and pitied the poor fellow from his heart, but had no conception that the poor patient he so commiserated with was in reality himself!'

Then there follows a description of the apparatus and instructions in its use.

*Biographical notes on Dr. William Guyton Atherstone:* Born at Sion Hill, Nottingham, on 29 May 1814. Accompanied his parents in 1820 to South Africa.

Educated at Grahamstown and later at Uitenborg.

Returned to Europe; obtained the M.R.C.S. (Eng.) in 1828 and the M.D. (Heidelberg) in 1829.

Returned to South Africa where he continued in general practice, including surgery, until his death in 1898.

Awarded the F.R.C.S. (Eng.) in 1861.

Keenly interested in botany, geology, railways, ostrich-farming, and telegraphic communications.

## FORTHCOMING INTERNATIONAL MEDICAL CONFERENCES

### 10TH INTERNATIONAL CONGRESS OF PAEDIATRICS—LISBON

The 10th International Congress of Paediatrics will be held in Lisbon from 9 to 15 September 1962 at the University of Lisbon. The programme will consist of 3 Plenary Sessions, at each of which a panel of 8 speakers will present papers. The official languages of the Congress are Portuguese, French and English. A social programme is being arranged, which will include an official banquet and dance, as well as a traditional bull fight.

All those interested in attending this Congress are requested to

register between August 1961 and January 1962, with the Secretariat of the Congress, Clínica Pediátrica Universitária, Hospital Santa Maria, Avenida 28 de Maio, Lisbon 4, Portugal.

### 8TH INTERNATIONAL CANCER CONGRESS—MOSCOW

The Eighth International Cancer Congress will take place in Moscow from 22 to 28 July 1961 at the Moscow State University, under the auspices of the International Union Against Cancer. The official languages of the Congress will be English, Russian and French. The Congress will be divided into the following groups:

1. Lectures by invited speakers.
2. Panel discussions on selected topics.
3. Section meetings where papers will be presented.

The topics selected for lectures are: Rôle of viruses in the origin of cancer; Biochemistry of cancer; Tumour biology; Experimental research and clinical oncology; New methods of cancer therapy; Radical cancer surgery; New methods in radiotherapy; and Cancer control.

Further information about the Congress may be obtained from the General Secretary, Soviet National Organizing Committee, Academy of Medical Sciences of the USSR, 14 Solyanka, Moscow, USSR.

## AMPTELIKE AANKONDIGING : OFFICIAL ANNOUNCEMENT

### SUID-AFRIKAANSE ONDERLINGE MEDIESE HULPVERENIGING : SOUTH AFRICAN MUTUAL MEDICAL AID SOCIETY

Lede van die Vereniging word nogmaals herinner aan die aankondiging en artikels wat in die uitgawes van die *Tydskrif* van 14 Januarie 1961, 28 Januarie 1961, 4 Februarie 1961, 11 Februarie 1961, 18 Februarie 1961 en 25 Februarie 1961 verskyn het. Die aandag van die lede word ook bepaal by die Omsendbrief wat aan al die lede van die Vereniging gestuur is, waarin lede versoek word om die Suid-Afrikaanse Onderlinge Mediese Hulpvereniging in kennis te stel dat hulle al hul pasiënte wat deur die Vereniging verseker is as private pasiënte beskou, wat persoonlik en direk verantwoordelik sal wees aan hul eie dokters vir die betaling van hul rekenings; en dat hulle nie bereid is om tjeks te ontvang wat deur die Vereniging aangebied word ter betaling van dienste wat aan dié pasiënte gelewer is nie. Die reaksie daarop is sover baie bevredigend, maar dit is wenslik dat lede wat dit nog nie gedoen het nie, sonder versuim die antwoordstrokies wat aan die omsendbrief geheg is, moet voltooi en pos.

As daar lede is wat om die een of ander rede nie die Omsendbrief ontvang het nie en wat dus nie hul antwoordstrokies ingevul het nie, word hulle versoek om te skryf aan die Sekretaris, Mediese Vereniging van Suid-Afrika, Posbus 643, Kaapstad.

L. M. Marchand
*Medesekretaris*

Plaza-gebou 28
Banksteeg
Pretoria
20 Maart 1961

Members of the Association are once more reminded of the notices and articles which have appeared in the issues of the *Journal* of 14 January 1961, 28 January 1961, 4 Februarie 1961, 11 February 1961, 18 February 1961 and 25 February 1961. Attention is further drawn to the Circular Letter which was sent to all members, in which members were requested to inform the South African Mutual Medical Aid Society that they regard all their patients who are insured with the Society as private patients, who will be personally and directly responsible to their doctors for their accounts; and that they are not prepared to accept cheques tendered by the Society in payment of accounts rendered to these patients. The response so far has been very satisfactory, but it is desirable that members who have not yet completed and posted the reply slips attached to the circular should do so without delay.

Members who, for some or other reason, have not received a copy of the Circular Letter and who, therefore, have not completed their reply slips, are requested to write to the Secretary, Medical Association of South Africa, P.O. Box 643, Cape Town.

L. M. Marchand
*Associate Secretary*

28 Plaza Building
Bank Lane
Pretoria
20 March 1961

## UNIVERSITEITSNUUS : UNIVERSITY NEWS

### UNIVERSITEIT VAN PRETORIA

By die Promosieplegtigheid op 17 en 18 Maart 1961 is die volgende grade en medaljes toegeken:

*Graad van Baccalaureus in Geneeskunde en Snykunde*

Albertyn, Christopher Charles (Posthuum)
Blignaut, Lourens Pieter Gildenhuys
Botha, Martha Elizabeth
Bothma, Jeremiah Jesaja
Brandt, Hans Dieter
Britz, Barend Marthinus Gerhardus
Buitendag, Bothma
Conradie, Christiaan Gert
De Lange, Louis Jacobus Kotze
Delport, Jacobus Petrus Daniel
De Mûelenaere, Magda Greta Beatrys Rufina
De Villiers, Guillaume Gerhardus
De Villiers, Jacob Isaac
De Wet, Jacobus Petrus
Du Toit, Louwrens Erasmus
Erasmus, Jacobus Abel
Gagiano, Carllo Andrias
Gerli, Ferdinando
Griesel, David Lambertus
Grobler, Cornelius Johannes
Haasbroek, Aletta Catharina
Hugo, Johannes Matthys (met lof in Interne Geneeskunde en Kindergeneeskunde)
Huygens, Henri Ghislain
Jaffe, Michael Myer
Jerling, Julius Coetzee
Joubert, Daniel Johannes
Joubert, Venard Pierre
Kmietsch, Clemens Maria (met lof in Obstetrie en Ginekologie)
Levin, Wilfred Jacob
Liebenberg, Nicolaas Dreyer
Meyer, Justus Wilhelm
Naudé, Marié Elizabeth
Oosthuizen, Pieter Cornelius
Peters, Johannes Wilhelm Karl
Pretorius, Helena Susanna Taillefort
Prinsloo, Coenraad Zacharias Joseph Viljoen
Said, Samuel Harry
Scholtz, Willem Christoffel
Smalle, Marc Juul Leo Geert
Snyders, Petrus Casparus Stephanus
Swanepoel, Jacobus Cornelius
Theron, Eduard Stanley
Van Dam, Alexander
Van den Berg, Pieter Johannes
Van der Lingen, Johann Christian Wilcocks
Van der Walt, Izak David
Van Drimmelen, Pieter
Van Konijnenburg, Dirk
Van Niekerk, Jacob Jozua
Van Schalkwyk, Hendrik Cornelius Hugo

Van Selms, Adrianus
Van Wyk, Abraham Johannes Joubert
Van Wyk, Matthys Johannes
Venter, Johan Christiaan
Vorster, Carl Theodorus
Wagener, Jac Jacobus Johannes
Weiss, Gabriel (met lof in Radiodiagnostiek en Radioterapie)
Zoete, Andre Emiel Cornelius

*Graad van Magister in Geneeskunde*

De Bruin, Micha (Anestesiologie)
Duncan, Harold James Mons (Chirurgie)
Du Toit, Carel Johannes (Oor- neus- en keelheelkunde)
Fontanive, Willy Werner (Radiologiese Diagnose)
Fourie, Rudolph Philippus (Anestesiologie)
Hough, Jacob (Interne Geneeskunde)
Minnaar, Willem Nicolaas (Interne Geneeskunde)
Venter, Izak David (Chirurgie)
(Vakke tussen hakies dui besondere-studierigting aan)

*Tweejarige Diploma in Radiografie*

Berkenbosch, Annie Magrietha
Brace, Marilyn Grace-Ann
Brody, Antoinette
De Villiers, Erica May
De Villiers, Maria Christina
Du Preez, Cornelia Catharina
Fourie, Berda Johanna
Gerber, Anna Dorothea (met lof)
Hinds, Hazel Maude
Leonard, Hannelé
Lubbinge, Catharina
Massyn, Hester
Nortjé, Sara Susanna
Otto, Anita
Richardson, Careen Rachel
Roettgers, Annette
Van Rooy, Frieda Marél
Van Vuren, Maria Magdalena Jansen
Viljoen, Emma Sophia
Visser, Johanna Hermina Catharina

*L. J. Te Groen-Medalje vir Obstetrie en Ginekologie*

Vir die finale jaar M.B., Ch.B.-student wat die beste in die vakke Obstetrie en Ginekologie presteer het: Kmietsch, Clemens Maria.

*Protea Holdings-Pryse*

(i) Vir die beste finale jaar M.B., Ch.B.-student in die vak Radiologie: Weiss, Gabriel.
(ii) Vir die beste finale jaar M.B., Ch.B.-student in die vak Interne Geneeskunde: Hugo, Johannes Matthys.
(iii) Vir die beste finale jaar M.B., Ch.B.-student in die vak Chirurgie: Van der Walt, Izak David.
(iv) Vir die beste finale jaar student in die Diplomakursus in Radiografie: Gerber, Anna Dorothea.

## ASSOCIATION NEWS

### FORMATION OF THE GERIATRIC SOCIETY OF SOUTH AFRICA

On Monday 20 March 1961 a meeting of members of the Medical Association was held in Cape Town to consider the formation of a group dealing with geriatric aspects of medicine.

Prof. J. F. Brock was in the chair, and more than 100 members attended. The first part of the meeting was devoted to a symposium on geriatrics. The speakers were Dr. B. Bronte-Stewart, Prof. J. H. Louw, Dr. H. Cooper, and Mr. J. N. de Klerk.

Dr. I. M. Hurwitz then proposed the following resolution: 'That this meeting of members of the Cape Western Branch of the Medical Association of South Africa decides to form a geriatric organization within the Medical Association of South Africa'. The resolution was unanimously adopted.

An *ad hoc* committee was formed to draw up a constitution and plans for the future of the organization. This committee met on Wednesday 12 April. Present were: Dr. I. M. Hurwitz (in the chair), and Drs. L. Blumberg, A. I. Goldberg, R. Katz, R. L. Retief, W. Silber, A. H. Tonkin, and I. Waynik.

It was decided that the name of the organization would be 'The Geriatric Society of the Medical Association of South Africa' and a constitution was adopted. Application has been made for the Society to be accepted as a group within the Medical Association of South Africa.

The first General Meeting of the Society will be held in Cape Town during Congress week in September, and Dr. J. H. Sheldon, who will be one of the visiting speakers at the Congress, will be invited to address the meeting.

All members of the Medical Association who wish to join the Society (annual subscription, R2) should write to: Dr. I. M. Hurwitz, 1014 Tulbagh Centre, Hans Strydom Avenue, Cape Town.

## IN DIE VERBYGAAN : PASSING EVENTS

*Mr. T. B. McMurray*, who has been the Hon. Registrar of the College of Physicians, Surgeons and Gynaecologists of South Africa since the appointment of the first Council of the College, has left for Australia. He has been appointed senior orthopaedic surgeon to the University of New South Wales and will be Director of the Orthopaedic Unit.

Mr. McMurray was the Hon. Secretary of the Steering Committee of the College before the first Council was elected and he gave a considerable amount of time and energy to building up the College, which owes a great deal of its success to him.

* * *

*Dr. T. B. McMurray*, ereregistrateur van die Kollege van Interniste, Chirurge en Ginekoloë van Suid-Afrika sedert die samestelling van die eerste Raad van die Kollege, het Suid-Afrika verlaat om hom in Australië te vestig. Hy is as senior ortopediese chirurg aan die Universiteit van New South Wales aangestel en sal die pos van Direkteur van die Ortopediese Afdeling beklee.

Dr. McMurray was die eresekretaris van die Reëlingskomitee van die Kollege totdat die eerste Raad gekies is. Hy het mildelik sy tyd en energie aan die opbou van die Kollege bestee en die mate van sukses wat die Kollege behaal het, is grootliks aan hom te danke.

*Dr. E. D. Cooper*, Medical Officer of Health, City of Cape Town, has left on an overseas visit. He will be returning at the end of August.

* * *

*World Mental Health Year* 1960. This special period of activity, which was initiated by the World Federation for Mental Health, will come to an end at the time of the Sixth International Congress on Mental Health in Paris from 30 August to 5 September 1961. A considerable number of new projects have been carried out during the period of World Mental Health Year in some 55 countries. In order to record these, the Secretariat of the Federation in London would appreciate being informed of any special activity undertaken *because* of World Mental Health Year, and which has not already been notified to them.

Information about the Sixth International Congress on Mental Health may be obtained from the Secretary, 19 Manchester Street, London, W.1.

* * *

*20th International Postgraduate Medical Course, Karlovy Vary, Carlsbad.* Since 1913, International Postgraduate Medical Courses have been held every second year in Karlovy Vary, the world-famous spa; and the next course will take place from 18 - 23

September 1961. The main subject of the course will be Diabetes mellitus; on which papers by leading research workers from Austria, Belgium, Bulgaria, France, Germany, Great Britain, Hungary, Italy, Roumania, Sweden, Switzerland, USA, USSR, Yougoslavia and Czechoslovakia will be presented. The official languages of the Course are English, French, German, Russian and Czech. Simultaneous translations in the official languages will be provided.

Excursions and social events will be arranged for all participants, and a special programme is being prepared for accompanying relatives. Further information may be obtained from the Czechoslovak Society of Physical Medicine, Albertov 7, Praha 2.

* * *

*The South African Institute for Medical Research, Johannesburg, Staff Scientific Meeting.* The next meeting will be held on Monday 8 May at 5.10 p.m. in the Institute Lecture Theatre. Miss Westwood will speak on 'Laboratory aspects of an influenza-vaccination study on gold miners'.

*Association of Physicians (M.A.S.A.), Cape Western Branch.* The next meeting will be held on Thursday 4 May 1961 at 2.30 p.m. at Medical School, Observatory, Cape. A panel of speakers consisting of Prof. J. Louw, Dr. S. Berman, Dr. P. E. Kottler, and Dr. M. A. de Kock will present papers on 'Cerebral and renal angiography', followed by a discussion period.

After a tea break, the meeting will resume on the subject of 'Treatment of malignant disease by means of radioactive isotopes and cytotoxic drugs'; and the speakers will be Dr. Anderson, Dr. Heyl, Dr. R. Mibashan, and Dr. R. Slome.

All those interested are invited to attend this meeting, and are requested to notify the Secretary, Dr. H. Muller, 812 Medical Centre, Cape Town.

* * *

*Mr. Lance Human*, surgeon, has given up his practice in Port Elizabeth, and is now practising in Uitenhage. Telephones: Rooms 27, residence 28.

*Dr. Lance Human*, chirurg, het getrek van Port Elizabeth na Uitenhage, waar hy nou praktiseer. Telefone: Spreekkamer 27, woning 28.

## NUWE PREPARATE EN TOESTELLE : NEW PREPARATIONS AND APPLIANCES

### MADRIBON SYRUP

Roche Products (Pty.) Limited announce the introduction of Madribon Syrup, and supply the following information.

*Description.* The long-acting sulphonamide, Madribon, sulphadimethoxine, has now become available in syrup form. It is a broad-spectrum antibacterial which has shown no toxicity and practically no side-effects. The syrup is very pleasantly flavoured and lends itself to accurate dosage by parents and nurses. Each teaspoonful (5 ml.) contains 0·25 g. of Madribon substance. Madribon syrup is intended for children and young adolescents.

*Indications.* The indications are for bacterial infections of any system; specifically where no culture and sensitivity tests can be performed.

*Dosage and Administration.* Madribon syrup is so designed that the dosage scheme can easily be memorized and risk of overdosage is thereby reduced. For moderate to severe infections the scheme is as follows:

Children aged 2 - 7 years—2 teaspoonfuls *statim*, and then 1 teaspoonful 24-hourly as maintenance dose.

Children aged 8 - 11 years—3 teaspoonfuls *statim*, and then 1½ teaspoonfuls 24-hourly as maintenance dose.

Children aged 12 - 14 years—4 teaspoonfuls *statim*, and then 2 teaspoonfuls 24-hourly as maintenance dose.

At these doses an adequate blood level of the active antibacterial substance, Madribon, is maintained slightly above optimum.

Further information may be obtained from the Scientific Department of Roche Products (Pty.) Limited, P.O. Box 6158, Johannesburg.

### VELBE

Eli Lilly and Company announce the introduction of Velbe, a new anticancer agent from a plant source, for treatment of generalized Hodgkin's disease and for a rare type of malignancy called choriocarcinoma, and supply the following information:

Velbe is the sulfate salt of vinblastine (VLB), an alkaloid extracted from the periwinkle, a garden shrub. Although it is found in many gardens, Lilly obtains the dried plant for processing in large quantities from the Far East.

Velbe has been studied in 300 patients with a variety of malignant diseases. It has been released for the treatment of Hodgkin's disease and choriocarcinoma. The evaluation of results in other cancers has not been completed.

The clinical trial is being expanded, and now includes more than 200 investigators throughout Canada, Australia, the United States, Europe, and Latin America.

Both the Lilly and Canadian clinicians reported particularly good effects against Hodgkin's disease. It was at the National Cancer Institute that the drug's usefulness in choriocarcinoma was first noted. The Lilly announcement summarizes the clinical studies of Hodgkin's disease as follows:

1. Significant tumour shrinkage and improvement in general physical condition were obtained in 31 of 34 patients. Of these 26 had previously failed to respond to other available methods of treatment.
2. Not less than 75% reduction in tumour masses occurred in at least half of the 34 cases.
3. To date, 23 patients placed on maintenance doses of Velbe have kept their improvement without relapse.
4. Many patients benefited by the drug and continue to be maintained in their improvement for periods ranging from several months to more than one year.
5. Several patients have lost, at least for the present, all evidence of Hodgkin's disease.

There is no evidence, however, that in any instance Velbe has 'cured' Hodgkin's disease or any other form of human cancer. No cancer is spoken of as possibly cured until there has been complete remission for 5 years.

At the National Cancer Institute, Velbe had produced antitumour effects in 7 of 10 patients with choriocarcinoma which failed to respond to other drugs. Five patients obtained clinical remissions of the disease. Four of the remissions have lasted from several months to a year or more.

Available only on prescription, the drug is administered by the physician by intravenous injection.

The activity of periwinkle extracts against cancers in animals was discovered independently by scientists of the Collip Research Laboratories at the University of Western Ontario, London, Ontario, Canada, and of the Lilly Research Laboratories, Indianapolis, USA.

# BOEKBESPREKINGS : BOOK REVIEWS

## AGEING

*Colloquia on Ageing*. Ciba Foundation. Vol. 5. The Lifespan of Animals. Edited by G. E. W. Wolstenholme, *O.B.E.*, M.A., M.B., M.R.C.P. and Maeve O'Connor, B.A. Pp. xii+324+46. 58 illustrations. 48s. net. London: J. & A. Churchill Ltd. 1959.

This volume contains the proceedings of the fifth Colloquium on Ageing held in April 1959. The contributors have produced a fascinating group of papers on such varied subjects as the life expectancy of offspring in relation to parental age, the lifespan of cattle and horses, arteriosclerosis in birds, longevity of fishes in captivity, factors influencing the lifespan of bees, and the biology of ageing in insects.

Some interesting conclusions were arrived at. In the past hundred years or so the peak in the age distribution of deaths in the general population has moved to a more advanced age (for men from 72 to 76 years and for women from 73 to 80) and the proportion of people attaining their allotted lifespan has increased for both sexes, from about 40% to about 70%. Arteriosclerosis has not been found in fishes, but pathological changes indicative of ageing do occur in other organs, e.g. cirrhosis and fatty changes in the liver and kidneys, haemochromatosis, and hypochromic anaemia. Observations on the accumulation of non-infectious lesions in rats in relation to age have shown that the lifespan of these animals is directly related to the age of onset of lesions of the major diseases of this species—chronic nephrosis and glomerulonephritis, myocardial degeneration, periarteritis, and pituitary adenoma. Data on human lesions, e.g. generalized arteriosclerosis, prostatic hypertrophy, coronary thrombosis, and carcinoma of the stomach, give similar curves to those of rats (plotted on the same age scale) except that the onset of the human diseases does not occur until many years later than is the case with rats.

Other interesting facts are the following: A maximum longevity of 7·5 weeks was found for honey bees. Female houseflies were found to have a mean longevity of 29 days and males a mean longevity of 17 days. The longest-lived freshwater fish is the sturgeon; the longest ever recorded was 151 years. The chairman points out in his summary that man, skunks, and porcupines are amongst the few animals who know how to survive into the period of senescence and remarks that 'the nature of this grouping probably has some moral significance'.

The book provides a valuable up-to-date account of developments in this important field.

In addition to the proceedings of the fifth Colloquium on Ageing this volume also contains a combined index to this and the previous four volumes. H.Z.

## HEALTH AND ILLNESS IN YOUNG CHILDREN

*Growing up in Newcastle upon Tyne*. A continuing study of health and illness in young children within their families. By F. J. W. Miller, S. D. M. Court, W. S. Walton and E. G. Knox. Pp. xxi+369. Illustrations. 25s. 0d. London: Oxford University Press. South African Office: Oxford University Press, P.O. Box 1141, Cape Town. 1960.

This book presents the facts of development and diseases of 847 children observed for 5 years (1947 - 1952).

Part I describes the families set against the social and economic background of Newcastle.

Part II deals with the housing, family, and medical care of the child.

Part III gives a detailed account of the illnesses of the first 5 years.

Part IV gives an estimation of the incidence of illness and defects in preschool children, calculated from the facts of the survey, and makes suggestions for further improvements in the health of the children.

There are many useful facts and figures presented in this volume that will prove of great interest to the practitioner and paediatrician. For example, only 11 children received no medical attention at all during the 5 years, 61% of infective illnesses were respiratory, and nearly all the serious and fatal infective illnesses occurred in the first year, except pneumonia. Behaviour problems occurred in only 55 children (6%). This study is to continue through the school years and may be regarded, as was its predecessor, 'A Thousand Families in Newcastle upon Tyne' (Oxford University Press, 1954), as a classic. J.D.L.H.

## COMPLICATIONS OF PREGNANCY

*Medical, Surgical and Gynecological Complications of Pregnancy*, By the staff of the Mount Sinai Hospital, New York City. Edited by Alan F. Guttmacher, M.D. and Joseph J. Rovinsky, M.D. Pp. x+619. Illustrated. $16.50. Baltimore: Williams & Wilkins Co. 1960.

This volume is an outcome of the commendable organization of the obstetric unit of the Mount Sinai Hospital. Ten specialty clinics (cardiac, pulmonary, hypertensive-renal, diabetic, haematological, neurological, psychosomatic, obstetric-gynaecological, endocrine, vaginitis, and varicose vein) function regularly within the framework of the obstetric department, to deal with the various intercurrent diseases that complicate pregnancy. Each clinic is conducted by a specialist in the field (physician, haematologist, surgeon, endocrinologist, etc.), as well as the obstetrician. This work is written by the team of specialists conducting these clinics, and is based on the material encountered at the Mount Sinai Hospital and supported by a most extensive reference to modern literature.

The result is a detailed and authoritative work dealing with practically every medical, surgical and gynaecological disease that may complicate the pregnant state—even conditions rarely encountered in pregnancy are dealt with fully, e.g. gunshot wounds of the pregnant uterus, Gaucher's disease, cancer of the thyroid, and diverticulitis. Particularly good chapters are those on the effect of radiation on the foetus, cancers during pregnancy, and pregnancy following plastic operations, but the standard of the entire work is high. It is a pity, however, that nephritis receives scant attention, and a few diseases such as parkinsonism, typhoid, and other acute fevers are omitted.

The publication can be confidently recommended, not only to those practising obstetrics, but also to physicians and surgeons. This book will undoubtedly become a valuable reference work for all who encounter the numerous medical, surgical, and gynaecological disorders that complicate pregnancy. In it will be found an abundance of information on these subjects as they affect the pregnant state, as well as a comprehensive list of the references that could be consulted. F.B.

## MEDICAL CARE

*The Demand for Medical Care*. A study of the case-load in the Barrow and Furness Group of Hospitals. By G. Forsyth, B.A. (Econ.) and R. F. L. Logan, M.D., M.R.C.P. Pp. 153. Illustrated. 7s. 6d. London, New York, Toronto: Oxford University Press. 1960.

This little book, which deals with a study of a group of Lancashire hospitals by the Nuffield Provincial Hospitals Trust, will be of special value to readers interested in public health and in hospital planning and administration. The authors apply a rational approach to the problem of determining whether the provision of beds is sufficient to meet present and future needs.

Of particular interest, in the light of the shortage of hospital accommodation in many parts of South Africa, is the frequency with which the admission of patients was found to have been 'clinically unnecessary'. In general medicine, over a quarter of the bed patients were receiving care which could equally well have been given in an outpatient department or at home. Such a finding underlines the potential value, for this country, of an expansion of home-nursing facilities and other neighbourhood health services aimed at reducing the need for hospital care. J.H.A.

## DERMATOLOGY

*Year Book of Dermatology*, 1959-1960. Edited by R. L. Baer, M.D. and V. H. Witten, M.D. Pp. 479. 59 figures. $9.00. Chicago: Year Book Publishers, Inc. 1960.

Nobody can complain about the quality of the abstracts in this Year Book, but the editors really should make some effort to escape from parochialism. Fifty per cent of the articles abstracted appeared in American journals, mainly the *A.M.A. Archives of Dermatology* and the *Journal of Investigative Dermatology*. The rest come from a limited range of journals from other countries that are, or ought to be, found in any university library. J.M.

## TOXAEMIA OF PREGNANCY

*The Aetiology and Arrest of Pre-Eclamptic Toxaemia with Early Ambulant Treatment*. By K. Douglas Salzmann, M.D., M.R.C.P. (Ed.), D.Obst.R.C.O.G. Pp. viii+69. 1 illustration. 10s. 6d. net. London: H. K. Lewis & Co. Ltd. 1960.

In this monograph the author discusses certain aspects of the aetiology, symptomatology, and treatment of pre-eclamptic toxaemia. He favours the 'toxin' theory, suggesting that the toxins arise from a damaged placenta. The primary damage is due to the reaction of the mother to hormones produced by the foetus or by a hydatidiform mole. The toxins have a vasopressor effect causing the hypertension.

According to the author the treatment is very simple—the prolonged use of reserpine with a maximum dose of 1·0 mg. twice daily, as soon as the diastolic blood pressure exceeds 84 mm. Hg. Treatment in bed and inductions become unnecessary.

His results are excellent, but the series (158 patients treated with reserpine) is too small for a serious claim to be made.

J.N.d.V.

## THE RETINA AND VISUAL PATHWAY

*Physiology of the Retina and Visual Pathway*. By G. S. Brindley, M.A., M.D. Pp. xi+298. Figures. 35s. net. London: Edward Arnold (Publishers) Ltd. 1960.

This sixth monograph of the Physiological Society is written by a specialist for specialists. It is a mine of accurate and up-to-date information on the chemical and neurological phenomena of vision. Experimental evidence for the principal theories is given in some detail and deductions from this evidence are closely reasoned. A chapter on the validity of subjective evidence, although concerned primarily with the visual sense, forms an important contribution to the whole problem of sensory experiments. The bibliography is comprehensive.

In spite of the author's praiseworthy attempt to keep the terminology simple, the book would be difficult reading for anyone with little previous knowledge of the physiology of vision. The reader, already versed in this speciality, who wishes to systematize his knowledge and bring it up to date, will find it a very useful addition to his library.

A.W.S.

# BRIEWERUBRIEK : CORRESPONDENCE

## A NEW METHOD OF TESTING DIABETICS

*To the Editor:* I was pleased to read about Dr. Brajtman's new method of testing diabetics in the Journal.[1] Today I had the opportunity of testing a diabetic patient of mine, first by the 'finger test' and secondly by 'Tes-tape' with urine. In both methods the colouration on the 'Tes-tape' was identical. The 'finger test' is an easier method, I agree, but I wonder if it will be effective in other diabetics as well. Like Dr. Bratjman, I am also interested in the mechanism of the reaction.

E. I. Bhorat

Lindley Street
Stanger
8 April 1961

1. Correspondence (1961): S. Afr. Med. J., 35, 279.

## A NEW METHOD OF TESTING DIABETICS

*To the Editor:* Dr. I. Brajtman brings forward in your correspondence columns[1] the interesting chance observation of Miller and Ridolfo which was published in *Diabetes* in the January/February number of 1960, under the title of 'The Skin-surface-glucose test: An aid in the diagnosis of diabetes mellitus'. The Pharmaceutical firm of Eli Lilly and Company, the makers of Tes-Tape (the urine-sugar analysis paper), in their *Physician's Bulletin* number 3, volume 25, page 50, give a very useful précis of the work done by Miller and Ridolfo as a result of their discovery and indicate the practical value of this skin-surface-test for physician and patient. In my experience I have found it particularly of value in the management of the patient whose hyperglycaemia is high, but whose renal threshold for sugar is also high.

Theodore James

Pinelands
Cape Town
3 April 1961

1. Correspondence (1961): S. Afr. Med. J., 35, 279.

## KNOTTING OF THE BOWEL

*To the Editor:* An article in the *Journal* for 8 April 1961[1] describes the condition of 'Knotting of the bowel' (Knoop in die derm). The author, in a perusal of the literature, failed to find any account of this condition, but it has been well described by Dr. N. J. Weinberg in the *Proceedings of the Mine Medical Officers' Association* (No. 336, vol. xxxviii September/December 1958).

I have dealt, personally, with three of these cases in Africans. In one case the primary loops were small bowel and sigmoid colon. In none of the cases, owing to gross distension of the intermediate loop, has it been possible to unravel the knot. The treatment under these circumstances is to section the bowel between clamps on the proximal aspect of the knot. The distal portion is then closed with a purse-string suture, and can then be withdrawn through the loops comprising the actual knot. Thereafter non-viable bowel or severely damaged bowel is resected and an end-to-end anastomosis performed.

The condition may resemble inter-mesenteric hernia when first seen, but in the cases described this has not been the cause. A search following resolution of the lesion failed to reveal any gap in the mesentery.

B. A. King

P.O. Box 87
Welkom
14 April 1961

1. Visser, J. D. (1961): S. Afr. Med. J., 35, 283.

## TELEGRAM TO PRIME MINISTER

*To the Editor:* I wish to record strong protest at the recent action of the South African Medical Council whereby this body submitted a telegram of welcome to the Prime Minister, Dr. Verwoerd, on the occasion of his return from London, having withdrawn South Africa's application for admission to the Commonwealth. Notice of such a telegram appeared in the lay press.

The circumstances which prevailed at the time were such that a telegram of this nature gave the impression that the Medical Council were in agreement with and sympathetic to the Prime Minister's actions. This in itself, appealing to a section only of the population, aligned the Medical Council with party-political expression. This is an affront to the professional status of this body and the medical profession as a whole.

The fact that the lay public does not readily distinguish the Medical Council from the Medical Association would lead to an impression that the former body represents the opinion and interest of the medical profession.

As a member of the Medical Association, I request the Association to record suitable protest.

Maurice Silbert.

7 Mimosa Arcade
Regent Road
Sea Point
7 April 1961

## TELEGRAM AAN DIE EERSTE MINISTER

*Aan die Redakteur:* Slegs hier sal 'n telegram aan die Eerste Minister beskou word as 'n politieke gebaar. Hy het teruggekeer as Eerste Minister van die Unie van Suid-Afrika en nie as leier van 'n politieke party nie.

As die telegram, wat verkeerdelik aan die lede van die Mediese Vereniging gekoppel word, werklik 'n belediging vir hulle is, is hulle nie die naam van Burgers van Suid-Afrika werd nie.

Ek sien geen nodigheid daarin dat die professie moet protesteer teen die telegram nie, en as huldeblyk aan 'n groot man was dit heel van pas.

J. A. Jooste

Posbus 213
Warden
11 April 1961